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ENDURANCE TRAINING AND DETRAINING; EFFECT on SYSTEMIC AND LOCAL MUSCLE PARAMETERS AND ON SERUM LIPIDS AND LIPOPROTEINS.

University — Université

ALBERTA

Degree for which thesis was presented — Grade pour lequel cette thèse fut présentée

Ph.D.

Year this degree conferred — Année d'obtention de ce grade

1980

Name of Supervisor — Nom du directeur de thèse

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ADAPTATIONS TO ENDURANCE TRAINING AND DETRAINING: CHANGES IN SYSTEMIC AND LOCAL MUSCLE PARAMETERS AND IN SERUM LIPIDS AND LIPOPROTEINS

by

© ALLISON ELIZABETH READY

A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
AND RESEARCH IN PARTIAL FULFILLMENT OF THE
REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF PHILOSOPHY

FACULTY OF PHYSICAL EDUCATION
EDMONTON, ALBERTA
FALL, 1980

THE UNIVERSITY OF ALBERTA

FACULTY OF GRADUATE STUDIES AND RESEARCH

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled ADAPTATIONS TO ENDURANCE TRAINING AND DETRAINING: CHANGES IN SYSTEMIC AND LOCAL MUSCLE PARAMETERS AND IN SERUM LIPIDS AND LIPOPROTEINS submitted by Allison Elizabeth Ready in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Physical Education.

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ABSTRACT

Twenty-six male volunteers ($\bar{x}$ age = 25.0 years) participated in an 18 week investigation of the effect of endurance training and detraining on selected physiological variables. Subjects were randomly assigned to exercise (n = 14) or control (n = 12) groups which were further subdivided on the basis of predicted $\dot{V}O_2$ max and designated as 'high fit' or 'lo fit'. The 9 week training program consisted of 4 thirty minute sessions per week on a bicycle ergometer at a training heart rate equivalent to 80% of $\dot{V}O_2$ max.

The response of systemic parameters and serum lipids and lipoproteins to training and detraining was monitored at 3 week intervals. Maximum oxygen intake and anaerobic threshold changed significantly ($p < 0.05$) with training and detraining. Submaximum heart rate decreased significantly with training; there was no significant change during the period of detraining. Maximum ventilation, maximum heart rate, and submaximal steady state oxygen consumption were not affected significantly by the program. There were no significant changes in total serum cholesterol, serum triglyceride, serum HDL-cholesterol, serum (VLDL + LDL)-cholesterol, and serum HDL-cholesterol/ total cholesterol in response to training or detraining.

Muscle biopsies of the vastus lateralis were taken from members of each group prior to training, post training, and post detraining. No significant differences existed between or within groups for muscle fiber type (I, IIa, IIb) or SDH activity. There were also no significant changes in body composition or diet throughout the program.

ACKNOWLEDGEMENTS

The author would like to thank her advisor, Dr. Art Quinney, for his encouragement and guidance throughout this study. Gratitude is also expressed to my committee members Dr. A. E. Wall, Dr. H. A. Wenger, Dr. M. Cottle and Dr. R. Macnab for their assistance and advice.

Thank you to Shirley Hilger whose technical expertise and sense of humour were invaluable in the preparation of this dissertation.

The author would also like to express her gratitude to Cheryl Luchkow for her diligence in typing the manuscript, and to Larry Coulson and Brian Pinchbeck for their capable assistance with computer programming and data analysis.

To my friends Cathy Macdonald and Ann Jones who helped keep everything in perspective: thank you for being there.

A special acknowledgement is extended to the individuals who served as subjects and whose enthusiasm, time, and effort were instrumental in the completion of this study.

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CHAPTER I

INTRODUCTION

Numerous physiological adaptations to endurance training have been documented, including elevations in maximal oxygen uptake ($\dot{V}O_2$ max), cardiac output ($\dot{Q}$), stroke volume (SV), and arterio-venous oxygen difference ($a\text{-}\bar{v}O_2\Delta$) (Rowell, 1974), as well as slower rates of glycogen depletion (Hermansen et al., 1967), lower blood and muscle lactate concentrations for a given workload (Ekblom et al., 1968; Saltin et al., 1971), and increased reliance on fat as an energy source (Hermansen et al., 1967; Hoppeler et al., 1973). Biochemical alterations in part responsible for these training effects include increases in the activity and concentrations of respiratory enzymes (Holloszy, 1967; Morgan et al., 1971) and enzymes involved in the oxidation of fatty acids (Molé et al., 1971). Changes in muscle fiber area (Taylor et al., 1978), subgroup distribution (Jansson et al., 1977), and mitochondrial size and structure (Gollnick and King, 1969) have also been recorded.

Longitudinal training studies using homogeneous subject populations have been scarce. Most research has involved cross-sectional comparisons between previously trained and untrained groups making generalization of results difficult. Lack of quantification of the training stimulus and control over extraneous variables has further hampered investigation in this area.

The response of endurance trained individuals to the cessation of training, or detraining, has received less attention. Early studies examined the effect of bed rest on aerobic fitness (Taylor et al., 1949;

Deitrick et al., 1948). Subsequent investigations have monitored cardiovascular changes in athletes following termination of the competitive season (Drinkwater and Horvath, 1972; Michael et al., 1972), and in sedentary subjects after the completion of short term training programs (Fringer and Stull, 1974; Smith and Stransky, 1976; Pedersen and Jorgensen, 1978).

Recent papers have dealt with the local adaptation of skeletal muscle to detraining. Changes in respiratory enzyme activity (Henriksson and Reitman, 1977; Orlander et al., 1977), fiber area (Houston et al., 1979) and composition, and ultrastructure (Orlander et al., 1977) have been reported. Common use of cross-sectional research models and lack of work quantification have been limitations in many of these studies.

Studies of the effect of endurance training on serum lipids have traditionally been directed toward cholesterol and triglyceride. Results have been inconsistent and are thought to have varied with changes in the exercise stimulus, diet, or morphology of the population studied. The recent development of techniques to fractionate serum cholesterol into component lipoproteins may enable better elucidation of changes in serum lipids with training. Discovery of inverse changes in high density lipoprotein (HDL) cholesterol and low density lipoprotein (LDL) cholesterol with regular exercise may explain the lack of significant findings in earlier studies (Lopez, 1976).

Little is known of the response of serum lipids to detraining. Decreases in serum triglyceride and cholesterol which resulted from one training program persisted during 8 weeks of detraining (Watt et al., 1972). Other investigators have found elevations of serum cholesterol to pretraining values shortly after the cessation of regular exercise (Rochelle, 1961; Cureton and Phillips, 1964). The effect of detraining on serum lipoproteins has not yet been established.

Knowledge of the magnitude and time course of physiological adaptations to training and detraining will assist in the design of more effective exercise programs. Advantages which may result from a better understanding of the detraining process include awareness of the effects of illness and injury on athletic performance. The establishment of optimal time lines to ensure peaking, and to stress various fitness components, as well as the development of satisfactory maintenance programs may also be enhanced by a greater understanding of detraining.

A negative relationship has been reported between HDL-cholesterol and coronary heart disease (CHD) (Berg et al., 1976). The discovery that HDL-cholesterol may increase with regular training has led to its proposal as an anti-atherogenic agent (Lopez, 1976). Examination of the serum lipoproteins during a controlled training regimen will increase the understanding of this mechanism. The study of lipoprotein changes during detraining may assist in the establishment of preventative and rehabilitative exercise programs for CHD.

The purpose of this study was to monitor the effect of endurance training and detraining upon selected systemic and local muscle parameters and to compare the magnitude and time course of any changes which occurred. The rate of decline in fitness gains responsible for maximal performance, as measured by $\dot{V}O_2$ max, and submaximal performance, as indicated by anaerobic threshold (AT), were also related to changes in variables representative of systemic and local changes.

The response of serum lipids and lipoproteins to chronic exercise and its termination has also been examined. An additional aim of this investigation was to distinguish between groups designated high in fitness (hi-fit) and low in fitness (lo-fit) in their adaptation to training and detraining.

Several limitations affected the findings of this investigation. Difficulties in methods of data collection , reliability of measurements, and problems of statistical design must be considered in interpretation of the results. Subject selection and control of diet and activity levels were further limitations of the study.

CHAPTER II

REVIEW OF THE LITERATURE

This chapter contains a review of the literature pertaining to the response of systemic and local muscle parameters to endurance training and detraining. A comparison of the time course and relative magnitude of changes in the above factors will also be presented. The effect of endurance training and detraining on serum lipids and lipoproteins is also reviewed.

SYSTEMIC ADAPTATION TO ENDURANCE TRAINING AND DETRAINING

Changes with Training

Improvements of the cardiovascular system with training have been extensively documented (Ekbolm, 1969; Saltin, 1969; Rowell, 1974; Clausen, 1977) and will be reviewed only briefly in this paper. Training adaptations during submaximal work include decreases in heart rate (Frick et al., 1967; Saltin et al., 1969), muscle blood flow (Klassen et al., 1970) and increases in stroke volume (Bevegard et al., 1963; Saltin et al., 1968). Cardiac output has been reported to decrease (Douglas and Becklake, 1968; Clausen et al., 1971), increase (Shepard and Simmons, 1972) and remain unchanged (Freedman et al., 1955) in response to work of this type.

The most noticeable effect of training on the circulatory system during exercise of maximal intensity is an increase in $\dot{V}O_2$ max (Astrand, 1952; Ekblom et al., 1968). Other changes include elevations of maximal cardiac output and stroke volume (Clausen, 1977). It is also believed that maximal muscle blood flow is increased by training

(Saltin et al., 1968; Clausen, 1977) although the extent to which this increase must be distributed to a larger muscle mass remains unclear. Maximal heart rate has been reported to decrease in response to regular exercise (Ekblom et al., 1968).

Changes in $\dot{V}O_2$ max with training are dependent upon the intensity, frequency and duration of the training program (Sharkey and Holleman, 1967; Wenger and MacNab, 1975) as well as the initial fitness of the participant (Saltin, 1969; Knuttgen et al., 1973). Improvements average from 15 to 30 percent after participation in short term training programs by previously unfit subjects (Saltin et al., 1977b). The greatest reported elevations in $\dot{V}O_2$ max have resulted from training which followed an extended period of bed rest (Saltin et al., 1968). Endurance athletes possess the highest recorded values for maximal oxygen intake (Saltin and Astrand, 1967).

The increase in $\dot{V}O_2$ max in previously sedentary young men exposed to training has been attributed to increments in both $\dot{Q}$ max and $a\text{-}\bar{v}O_2\Delta$ max (Rowell, 1962; Saltin et al., 1968; Ekblom et al., 1968). Studies of women and middle aged men, however, ascribed elevations in maximal aerobic capacity solely to increments in $\dot{Q}$ max (Hartley et al., 1969; Kilbom, 1971). Recent evidence suggests that the nature of the exercise stimulus may affect the cardiovascular adaptation. Long term training programs, and programs of relatively high intensity, have demonstrated significant increases in $a\text{-}\bar{v}O_2\Delta$ max as well as $\dot{Q}$ max in women (Cunningham and Hill, 1975; Cunningham et al., 1979).

Increments in $\dot{Q}$ max from 8 to 13 percent have been reported with short term exercise programs (Ekblom et al., 1968; Kilbom, 1971; Cunningham and Hill, 1975). Clausen (1977) examined the results of several previous studies and found an average increase of 12% in $\dot{Q}$ max following training of from 4 to 16 weeks (Rowell, 1962; Saltin et al., 1968; Hartley et al., 1969; Kilbom and Astrand, 1971; Gleser, 1973).

It has generally been accepted that increases in $\dot{Q}$ max are the result of elevations in stroke volume (Saltin, 1969; Cumming, 1975). The absence of change, or slight reduction, found in maximal heart rate with training is the basis for this belief. Clausen (1977) has stated that this response does not imply that the increase in $\dot{Q}$ is brought about solely by central circulatory adaptations. After examination of the results of several studies that measured both mean arterial pressure and $\dot{Q}$ he has suggested that a reduction in total peripheral resistance may occur with training, possibly augmenting maximal flow capacity and increasing $\dot{Q}$ max. Other studies have found no change in maximal muscle blood flow with training (Grimby et al., 1967). Recent investigations of capillary supply in humans may help clarify this issue (Brodal et al., 1977; Andersen and Henriksson, 1977b).

The high $\dot{V}O_2$ max possessed by athletes is largely the result of an increased SV (Saltin and Astrand, 1967; Ekblom, 1968). Values of 42 ml and 210 ml have been reported for cardiac patients and athletes respectively (Ekblom and Hermansen, 1968; Rowell, 1974). Average SV max in a normal untrained male is approximately 100 ml (Cumming, 1975). Increases in maximal stroke volume have also been demonstrated during longitudinal training programs. Ekblom et al. (1968) reported an elevation of 20% from 122 ml to 146 ml, after 16 weeks of exercise by previously untrained subjects. Other studies have reported increases of between 13 and 28 percent (Ekblom, 1969; Kilbom, 1971; Cunningham and Hill, 1975). An increase of 62%, from 74 ml to 120 ml, occurred in subjects who were trained following a period of bed rest (Saltin et al., 1968).

Changes with Detraining

Early research focusing on the effects of detraining on the cardiovascular system involved previously fit subjects confined to bed rest (Deitrick et al., 1948; Taylor et al., 1949). Following 20 days of immobilization by 5 men Saltin et al. (1968) reported declines of 27% and 26% in $\dot{V}O_2$ max and $\dot{Q}$ max respectively. It has also been demonstrated that inactivity, and not the time spent in a recumbent position, leads to deconditioning. Confinement of subjects to a space cabin simulator in one study resulted in significantly decreased maximal oxygen consumption and performance time on a treadmill test, as well as increased heart rates at a given submaximal workload (Lamb et al., 1964). Birkhead (1963) attributed a significant decrease in $\dot{V}O_2$ max following 6 weeks of bed rest to lower hemoglobin concentrations.

Interest has also been shown in the establishment of exercise programs that will optimally maintain fitness gains following training. Recommended exercise frequencies for retention of fitness include three times per week (Brynteson and Sinning, 1973), every third day (Roskamm, 1967) and once per week (Kriesseg, 1969; Chaloupka and Fox, 1975). Further knowledge of the time course of changes with detraining may assist in better understanding of the retention process.

Few studies have investigated the effect of the training stimulus on subsequent detraining. Case (1971) found little difference in the rate of detraining between subjects who had trained twice per week and those who had trained four times per week. In a two month study employing three types of interval training Knuttgen et al. (1973) found no significant difference in physiological parameters between groups after 8 months of detraining. It was also concluded by Smith and Stransky (1976) that the nature of the training program had little effect on the detraining process. Loss of training gains was equal whether subjects had participated in running or cycling. Applegate and Stull (1969) however, state that comparisons between maintenance of gains in strength, muscular endurance, and endurance may be misleading.

Intensity and duration of the training regimen must also be considered relevant to the detraining process. These factors influence the amount of endurance gained and consequently the amount available to be lost. In a study of college women who had trained for 8 weeks, Schuble (1972) found no changes in cardiovascular parameters following 5 or 10 weeks of detraining. It was suggested that the initial training stimulus had not been of sufficient intensity to cause changes to occur with its cessation.

Evert (1972) followed the detraining of female track athletes for a 7 week post season period. No significant changes were found in several cardiovascular measures at a standard workload and it was concluded that submaximal exercises may not be sufficiently demanding to demonstrate detraining effects. Applegate and Stull (1969) also reported no changes with detraining. Rest periods of 2, 4, and 6 weeks following a 6 week conditioning program failed to elicit different effects on parameters of circulatory fitness. Similar results were reported by Triguero (1965) in a study of basketball players. During 10 weeks of detraining no significant changes in the heart rate response to a step test were found.

The time course of the decrement of cardiovascular parameters with detraining is of great practical importance. Michael and Gallon (1959) found that increases in heart rate recovery from a step test, gained through training, had returned to pretraining levels by 10 weeks. No additional changes resulted from 30 weeks of detraining. A detraining period of 4 weeks was found sufficient to return recovery heart rates after a step test to pretraining values by Hammer (1965). Changes that occurred in heart rate responses were greatest during the initial 7 weeks of detraining by female track athletes (Michael et al., 1972). Further significant increases had also occurred by 23 weeks.

The response of $\dot{V}O_2$ max to periods of detraining has also been investigated. Although a rapid decline of fitness gains was reported in middle aged men after 8 weeks of detraining, not all of the gains in $\dot{V}O_2$ max were lost (Cureton and Phillips, 1964). Following 7 weeks of interval run training Tanzi (1967) found that subjects had reverted to pretraining values for maximal oxygen intakes within 4 weeks. Fardy (1969) also found a rapid deterioration of maximum aerobic power. He recorded a significant decrease in $\dot{V}O_2$ max by the fifth week of detraining after a 10 week program of soccer training.

Retention of gains in performance of endurance activities have also been studied. Howells (1965) found 23 female subjects retained 30.1% of increased performance time on the bicycle ergometer after 1 year of detraining which followed an 8 week training program.

Significantly greater retention of performance capacity than $\dot{V}O_2$ max was also found after 2 weeks of detraining by Ready (1977).

Recent studies of the effects of detraining on cardiovascular fitness have involved the monitoring of athletes following the termination of their competitive season, and of previously sedentary subjects after the completion of short term training programs. Michael et al. (1972) assessed 10 girls from a high school track team 1, 3, 5, 7, and 23 weeks post training. Tests used were a 3 minute step test and a treadmill run. Although no significant changes were measured in $\dot{V}O_2$ max or oxygen debt during the detraining period, submaximal heart rate had increased significantly after 3 weeks. The authors concluded that the changes that did occur were greatest after 7 weeks.

The physiological effects of detraining on athletes following the track season were also studied by Drinkwater and Horvath (1972). $\dot{V}O_2$ max of 7 female track competitors was evaluated during the last month of the season and again 3 months after the cessation of formal training. It was concluded that 3 months of detraining had reduced the cardiorespiratory fitness of these subjects to levels found in untrained girls of the same age. $\dot{V}O_2$ max fell from 47.8 $ml.kg.min^{-1}$ to 40.4 $ml.kg.min^{-1}$ during the period of no training.

Fringer and Stull (1974) trained 44 women for 10 weeks in order to monitor the ensuing detraining process. Training consisted of 'all-out' rides on the bicycle ergometer twice a week. $\dot{V}O_2$ max, total work output, and maximal ventilation volume were retested 5 and 10 weeks following the program. Thirty-two percent of the gain in maximal aerobic capacity was retained after 5 weeks of detraining. This fell to 19% after 10 weeks. Improvements in pulmonary ventilation and work

TABLE 2.1

EFFECT OF TRAINING AND DETRAINING ON MAXIMAL OXYGEN INTAKE (FEMALES)

Study	Training	$\dot{V}O_2$ max(ml.kg.min^{-1})			Wks. of Detraining	% Change	% Retention
		pre-train (t_1)	post-train (t_2)	post-detrain (t_3)		t_2-t_3	t_1-t_3
PEDERSEN & JORGENSON (1978)	n = 6 2x/wk., 7 wks. bike	41.5	46.7	43.8	7	-6.21	44.2
READY (1977)	n = 7 3x/wk., 6 wks. bike	38.6	41.1	39.2	2	-4.63	24.0
	n = 2 3x/wk., 6 wks. bike	42.68	50.45	42.80	8	-15.17	1.5
FRINGER & STULL (1974)	n = 44 2x/wk., 10 wks. bike	33.77	45.76	37.57	5	-17.9	32
		34.09	47.28	36.64	10	-22.5	19.3
DRINKWATER & HORVATH (1972)	n = 7 track season	-	47.8	40.4	21	-15.5	-

output were also retained to some degree after the detraining process.

A rapid return of cardiovascular variables to pretraining levels was found by Smith and Stransky (1976). Sixteen female subjects trained for 7 weeks on the bicycle ergometer. After 7 weeks of detraining submaximal heart rates and ventilation volumes had increased significantly. Two months was felt to be sufficient time for the loss of training gains resulting from either a long or short training program.

Similar conclusions were reached by Pedersen and Jorgenson (1978) in a study of 6 women. Subjects participated in a program of intense endurance training on the bicycle ergometer twice a week for 7 weeks. Maximal oxygen uptake increased by 13.8% during the training period. The average response was a relatively linear gain of 1.4 to 2.0 percent per week. At the end of the detraining period $\dot{V}O_2$ max was not significantly different from the initial value.

Few studies have examined the mechanisms which control cardiovascular changes with detraining. A recent investigation of the coronary vasculature of dogs during deconditioning suggests that central modification of the cardiovascular system is responsible for a large proportion of changes with training and detraining (Wyatt and Mitchell, 1978). Following 12 weeks of treadmill training a group of dogs detrained for 6 weeks. Significant reduction occurred in the cross-sectional area of the circumflex artery and in myocardial capillary density. Similar findings have been reported by Leon and Bloor (1968, 1976) in studies of rats.

The activity of cardiac actomyosin ATPase has been shown to decrease following detraining in rats (Malhotra et al., 1976; Scheuer et al., 1976). The authors suggest that this may represent an important controlling factor responsible for the increased end systolic volume in trained hearts. Reductions in SV max with detraining may result from changes in the activity of this enzyme.

LOCAL MUSCLE ADAPTATION TO ENDURANCE TRAINING AND DETRAINING

Changes with Training

Changes in $a\text{-}\bar{v}O_2\Delta$ max with training are thought to account for approximately 50% of the increase in $\dot{V}O_2$ max in previously sedentary young men (Rowell, 1974). Depending on the intensity and duration of training $a\text{-}\bar{v}O_2\Delta$ max has also been reported to increase in women (Cunningham and Hill, 1975; Cunningham et al., 1979). Endurance athletes possess relatively high $a\text{-}\bar{v}O_2\Delta$ max. Values of 190 ml/liter and 156 ml/liter have been reported for athletes by Astrand et al. (1964) and Ekblom and Hermansen (1968). Average values in previously untrained young men approximate 120 ml/liter (Ekblom, 1969).

Possible mechanisms responsible for this adjustment include redistribution of a greater fraction of the $\dot{Q}$ to working muscle, or greater extraction of O_2 by the working muscle. It is unlikely that the first possibility plays a significant role in the increase in $a\text{-}\bar{v}O_2\Delta$ max (Rowell, 1974). Increased extraction of O_2 by muscle, as the result of several biochemical and structural adaptations to training may account for 50% of the increase in $\dot{V}O_2$ with training (Holloszy, 1975).

The metabolic and contractile properties of muscle fibers act as important determinants of physiological performance capacity (Karlsson et al., 1978). Changes in fiber subgroup populations with training may enhance the oxidative capacity of muscle thereby increasing $\dot{V}O_2$ max. Studies have consistently found a large proportion of type I fibers in endurance athletes (Gollnick et al., 1972; Kiessling et al., 1974; Costill et al., 1976a,b; Jansson and Kaijser, 1977). High correlations have also been found between % ST fibers and $\dot{V}O_2$ max (Orlander et al.,

TABLE 2.2

PERCENTAGE DISTRIBUTION OF FIBER TYPES IN VASTUS LATERALIS OF ATHLETES

Study	Population	Fiber Type				
		I	II	IIA	IIB	IIC
Ingjer (1978)	untrained	42.2	57.6	33.2	18.7	5.7
	P.E. students	50.9	49.1	31.6	11.9	5.6
	endurance trained	67.6	32.4	20.1	6.1	6.2
Houston et al. (1979)	distance runners	64.2	-	35.7	-	-
Jansson and Kaijser (1977)	untrained	53.9	46.1	32.2	13.0	.9
	orienteers	68.1	31.9	24.4	3.3	4.2
Prince et al.* (1976)	untrained	35.5	-	38.1	26.2	-
	distance runners	44.3	-	39.7	4.5	-
	weight lifters	45.0	-	10.5	33.3	-
Costill et al. (1976b)	untrained	57.7	42.3	-	-	-
	middle distance	61.8	38.2	-	-	-
	distance	79.0	21.0	-	-	-
Costill et al. (1976a)	untrained	52.6	47.4	-	-	-
	sprinters	24.0	76.0	-	-	-
	middle distance	51.9	48.1	-	-	-
	distance	69.4	30.6	-	-	-
	jumpers	46.7	53.3	-	-	-

*SO, FOG and FG.

1977; Bergh et al., 1978).

The ratio of type I to type II fibers is fairly constant in untrained subjects and is usually near 50:50 (Costill et al., 1976a; Green et al., 1979). Reported values range from 36:64 (Gollnick et al., 1972) to 58:42 (Costill et al., 1976a). High ranges in fiber population are reported for all groups (Edstrom and Ekblom, 1972; Gollnick et al., 1972).

The close relationship between success in endurance events and fiber distribution does not necessarily indicate that a change occurs in fiber types with training. Fiber type may be determined genetically and athletic success the result of a natural selection process. Although the oxidative capacity of fibers is known to change with training there is little evidence of a change in myosin ATPase activity following short term exercise programs (Andersen, 1975; Taylor et al., 1978; Orlander et al., 1980). Jansson and Kaijser (1977) found the proportions of type I muscle fibers in élite orienteers similar in the trained muscles of the leg and untrained muscles of the arm suggesting that fiber distribution is genetically determined and not the result of extreme endurance training.

Recent research indicates that there may be a gradual transition between type I and type II fibers during long term training. Karlsson et al. (1978) suggest that the FTc fiber may be an intermediate fiber between FTa and ST fibers. One study of 4 long distance runners following an 11 week period of anaerobic training and an 18 week period of aerobic training supports this theory (Jansson et al., 1978). Runners were found to have a significantly lower proportion of type IIC fibers (12% vs. 1%) after the period of anaerobic training. An

TABLE 2.3

EFFECT OF TRAINING ON PERCENTAGE DISTRIBUTION OF MUSCLE FIBERS IN HUMAN VASTUS LATERALIS

STUDY	TRAINING PROGRAM		FIBER TYPE*				
			I	II	IIA	IIB	IIC
Jansson et al. (1978)	n = 4	(Post aerobic)	69	31	20	10	1
	runners	(Post anaerobic)	52	48	18	18	12
Taylor et al. (1978)	n = 16	♂	45(45)	55(52)	-	-	-
	n = 21	♀					
	16 weeks endurance bike		49(51)	51(49)	-	-	-
Andersen and Henriksson (1977a)	8 weeks endurance bike		36(42)	64(58)	36(42)	20(13)	4.7(2.1)
Andersen and Henriksson (1977b)	8 weeks endurance bike		41(43)	59(57)	37(42)	19(14)	2.6(1.2)
Henriksson and Reitman (1976)	7-8 weeks endurance bike		50(50)	50(50)	-	-	-
Andersen (1975)	7 weeks endurance		48(51)	52(49)	-	-	-
Kiessling et al. (1974)	10 week endurance		47(48)	53(52)	-	-	-
Gollnick et al. (1973)	20 weeks endurance bike		32(36)	68(64)	-	-	-

*pre and (post) training

increased ratio of IIB:IIA fibers was also recorded in 3 subjects. The authors concluded that a transition from type I to IIC fibers, and from type IIC to type I fibers may result from anaerobic and aerobic training respectively.

Several studies have reported changes in fiber subgroup pattern with training. Prince et al. (1976) studied fiber distribution in distance runners, weightlifters and untrained subjects. Eighty-four percent of fibers in the runners and 55% in the lifters were oxidative (SO and FOG). The proportion of SO fibers was not significantly different and FOG fibers accounted for the discrepancy. While the distance runners possessed 4.5% FG fibers and 39.7% FOG fibers the lifters had 33.3% and 10.5% respectively.

In a study comparing élite orienteers with controls the quantitative relationship between IIA and IIB fibers was changed in favor of type IIA in the former group (Jansson and Kaijser, 1977). An increase in type IIC fibers was also noted and is further evidence of adaptation towards increased oxidative capacity. No change in distribution of type I fibers was noted in an 8 week bicycle endurance study (Andersen and Henriksson, 1977a) although an increase in the percentage of IIA fibers, and a corresponding decrease in the percentage of IIB fibers occurred. Type IIA fibers rose from 65% to 75% of the total concentration of type II fibers. Green et al. (1979) discovered similar alterations in a study of élite ice hockey players. Although the athletes were not significantly different from untrained controls in the percentage of ST fibers pre or post season changes were evident in the FT fiber subgroups. A reduction in FTb fibers from 12.2% to 2.9% and an increase in FTa fibers from 38.0% to 42.5%

occurred during the 6 month season.

Quantitative increases in several enzymes may also partially account for the changes in $\dot{V}O_2$ max with training. There is a positive relationship between the ability of a muscle to perform work and the activity of its respiratory enzymes (Lawrie, 1953). Significant correlations have been found between endurance, as reflected in the duration of a run to exhaustion, and the concentration of cytochrome C (CYT C) and citrate synthase (CS) in rat gastrocnemius (Fitts et al., 1975). Several recent studies have examined the effect of endurance training on mitochondrial respiratory chain enzymes (Holloszy et al., 1970; Morgan et al., 1971; Henriksson and Reitman, 1976), citric acid cycle enzymes (Holloszy et al., 1970; Orlander et al., 1977), and enzymes evolved in the oxidation of fatty acids (Jansson and Kaijser, 1977) and ketone bodies (Winder et al., 1974).

The activity of succinate dehydrogenase (SDH) is commonly used to indicate the involvement of the citric acid cycle in energy production. This enzyme catalyzes the oxidation of succinate to fumarate with the concomitant production of $FADH_2$ (Stryer, 1975). Unlike other enzymes of the citric acid cycle SDH is an integral part of the inner mitochondrial membrane and is directly linked to the electron transport chain.

The study of SDH has helped to elucidate adaptations which take place in enzyme activity with training. These changes are specific to local muscle groups (Benzi et al., 1975). Gollnick et al. (1972) reported the highest enzyme activity in muscle groups used by various athletes. SDH activities of the deltoid muscles of canoeists and swimmers were 2.2 and 2.4 times as great as those of untrained subjects.

In another study élite orienteers were found to have greater SDH activity in the gastrocnemius than the vastus lateralis (Jansson and Kaijser, 1977) reflecting the greater use of the former in level running (Costill et al., 1974).

Several comparative studies have noted elevated activity of SDH in endurance athletes (Gollnick et al., 1972; Costill et al., 1976b). The proportion of type I fibers was found to be generally high in these individuals and the possibility exists that the enzyme pattern may result from fiber distribution and not a training stimulus. This was refuted by Hansson and Kaijser (1977) who found the same proportion of type I fibers in untrained subjects as in élite orienteers although oxidative enzymes activity was considerably lower in the former group. Other studies have reported low correlations between % ST fibers and SDH activity (Costill et al., 1976b; Foster et al., 1978). A large part of the enhanced SDH activity which results from training may be explained by increased oxidative capacity of FT fibers.

The activity of many respiratory enzymes has been found to double with training. Holloszy (1967; et al., 1970) found a 2 fold increase in the activities of SDH, NADH dehydrogenase, NADH Cytochrome C reductase, succinate oxidase and cytochrome oxidase in rats after a program of treadmill running. Many studies have reported changes in SDH activity in man after completion of short term training programs. These changes are closely related to the intensity and duration of the training stimulus which explains the variance in findings between studies (Benzi et al., 1975; Fitts et al., 1975).

One endurance training program of five months duration elicited a marked increase in SDH activity (Gollnick et al., 1973). Subjects

TABLE 2.4

SDH ACTIVITY OF VASTUS LATERALIS IN ATHLETIC POPULATIONS (MALES)

STUDY	ACTIVITY	n	SDH ACTIVITY (μmoles x g^{-1} x min^{-1})
Gollnick et al. (1972)	untrained	26	4.4
	bicyclists	4	11.0
	canoeists	4	5.8
	runners	8	6.4
	swimmers	5	7.6
	weightlifters	4	3.0
	orienteers	11	5.7
Costill et al. (1976a)	untrained	11	7.4
	sprint runners	2	12.9
	middle distance runners	7	14.8
	distance runners	5	16.6
	jumpers	2	9.4
	javelin throwers	3	4.8
	shot, discus	4	4.3
Jansson & Kaijser (1977)	untrained	69	10.4
	élite orienteers	8	14.8
Houston et al. (1969)	distance runners	6	14.7

trained on the bicycle ergometer for 1 hour per day, 4 days per week at a load requiring from 75 to 90 percent of maximal aerobic power. Mean activity of SDH increased by 95%, from 4.65 to 9.06 μmoles x g^{-1} x min^{-1}. Eriksson et al. (1972) found a mean increase of only 29% in SDH activity of 11 to 13 year old boys who had trained regularly. Subjects pedalled 3 times a week for at least 20 minutes a session during the 6 week program. The discrepancy in intensity and duration of training between these two studies may account for the inconsistent results. Recent investigations of the effect of training on SDH activity are summarized in Table 2.5.

A relationship has been found between fiber recruitment and oxidative adaptation (Henriksson and Reitman, 1976). Exercise requiring less than $\dot{V}O_2$ max resulted in recruitment of type I fibers, whereas in exercise of greater intensity both type I and type II fibers were recruited continuously from the start of exercise (Gollnick et al., 1974). Henriksson and Reitman had subjects participate in either an 8 week interval training or continuous training program on the bicycle ergometer. SDH activity increased significantly (32%) only in type I fibers in the continuous group, and only in type II fibers (49%) in the interval group.

It has been proposed that $\dot{V}O_2$ max is limited by the oxidative capacity of mitochondria in skeletal muscle as well as the capacity of the oxygen transport system (Hoppeler et al., 1973). High correlations found between $\dot{V}O_2$ max and the volume density of central mitochondria, the surface of mitochondrial cristae, and the ratio of mitochondrial volume to myofibrillar volume support this proposal. Enzymes of oxidative phosphorylation, the citric acid cycle, and beta oxidation are located on the inner mitochondrial membrane and mitochondrial matrix. Increased activities of these mitochondrial bound enzymes following training suggest the occurrence of corresponding structural changes in the mitochondria.

In an early study by Gollnick and King (1969) it was shown that both the number and size of mitochondria increased in trained rats. Cristae concentration was also found to be more dense in the rats who

TABLE 2.5

EFFECT OF TRAINING ON SDH ACTIVITY OF VASTUS LATERALIS (MALES)

STUDY	TRAINING PROGRAM	SDH ACTIVITY (μmoles x g^{-1} x min^{-1}) PRE	POST	% CHANGE
MORGAN et al. (1971)	n = 10 4 wks. endurance bike	4.2	5.7	35.7
GOLLNICK et al.	n = 6 4x/wk., 20 wks. endurance bike	4.65	9.06	95.0
ERIKSSON et al. (1972)	n = 8 (age 11-13) 3x/wk., 6 wks. endurance bike	5.43	7.01	29.1
HENRIKSSON & REITMAN (1976)	n = 9 3x/wk., 7 to 8 wks. continuous training (bike)	10.8	13.5	22.0
	Interval training (bike)	9.1	11.6	27.5
ANDERSEN & HENRIKSSON (1977)	n = 5 4x/wk., 8 wks. endurance bike	8.6	12.2	41.8
HENRIKSSON & REITMAN (1977)	n = 13 4x/wk., 8 to 10 wks. endurance bike	9.5	12.54	32.0
TAYLOR et al. (1978)	n = 16 5x/wk., 16 wks. endurance bike	5.12	11.23	11.2

ran on the treadmill daily for 10 weeks than in controls. A study of humans utilizing a 1 month bicycle ergometer program found a significant change in mitochondrial volume (Morgan et al., 1971). Kiessling et al. (1971) reported increased numbers of interfibrillar and perinuclear mitochondria in humans who had participated in 14 weeks of training.

Values had doubled after 28 weeks. It was also found that élite athletes did not possess significantly more mitochondria than the previously sedentary subjects.

Size of mitochondria was not altered by the 7 month program. Athletes, however, were found to have significantly larger values than the sedentary subjects both pre and post training. It was concluded that prolonged moderate exercise led to an increase in the number of mitochondria and little increase in size, and that heavy training resulted in little further increase in number and a large increase in size. Comparison between élite orienteers and untrained subjects revealed similar findings (Hoppeler et al., 1973). Volume density of central and peripheral mitochondria, ratio of central mitochondrial volume to volume of myofibrils, surface of central mitochondria and mitochondrial cristae, and the size of the central mitochondria were all significantly greater in the trained orienteers.

Gains in aerobic power which result from endurance training are often accompanied by an increase in capillary supply (Andersen, 1975; Ingjer and Brodal, 1978). A linear relationship has been found between $\dot{V}O_2$ max and the average capillary number around each fiber (Ingjer, 1978). Changes in oxidative enzyme activity (Costill et al., 1976) and mitochondrial content (Hoppeler et al., 1973) also correspond well to adaptations in capillary supply.

It has been suggested that high capillary density in animals may enhance oxygen diffusion in muscle (Krogh, 1969). Improvements in physical performance following endurance training might then be partially attributed to changes in capillary density. Several studies have reported no change in capillary density with training. In an early report by Saltin et al. (1968), it was determined that although there was a large increase in $\dot{V}O_2$ max, there was no change in the size of capillaries or capillary density after training. Hermansen and Wachtlova (1971) compared capillary supply in 8 untrained and 7 well trained men. No significant difference was found between groups either at rest of after maximal exercise. A larger capillary to fiber ratio in the trained subjects was attributed to fiber area, and as a result there were no differences in diffusion distance. A study of capillary formation in rats following 4 weeks of endurance swimming also found no proliferative activity (Ljungqvist and Unge, 1977).

Recent studies in humans have documented changes in capillary supply with training. It is possible that earlier studies were hampered by poor measurement techniques. In a cross-sectional study of 12 untrained and 11 endurance trained subjects Brodal et al. (1977) concluded that one possible mechanism by which oxygen extraction in the muscle could be increased was by changes in capillary density. Capillary to fiber ratio, the number of capillaries surrounding each fiber, and capillary density were greater in the trained subjects by approximately 40%. $\dot{V}O_2$ max was also elevated by 40% in these subjects. Comparison of capillary supply in 5 well trained and 6 untrained women resulted in similar findings (Ingjer and Brodal, 1978). Capillary to fiber ratio, the number of capillaries around each fiber, and capillary density were greater by 52%, 45%, and 34% respectively.

In a longitudinal study significant increases occurred in capillary density and capillary to fiber ratio in 3 subjects who had followed a 7 week bicycle training program (Andersen, 1975). Significant increased in $\dot{V}O_2$ max, capillary density, and capillary to fiber ratio also occurred in 5 subjects who had trained for 8 weeks at 80% of maximal aerobic capacity (Andersen and Henriksson, 1977b).

Type I and type IIA fibers have significantly greater capillary contacts than type IIB fibers (Andersen, 1975). Capillaries around each fiber type appear to increase linearly with training. An increase of from 10% to 13% in capillaries around each fiber type has been found following an endurance training program (Andersen and Henriksson, 1977b).

A study examining the effect of low frequency activation of fast muscle in rabbits related the metabolic changes in muscle to an increased capillary supply (Brown et al., 1976). After only 4 days of stimulation there were increases in capillary density and capillary to fiber ratio indicating actual growth of capillaries. These changes, together with a decrease in mean fiber diameter, resulted in a shorter diffusion distance for oxygen. Changes in oxidative enzyme capacity occurred following the alteration in capillary supply.

Changes with Detraining

Studies of the effect of local immobilization and disuse on skeletal muscle preceeded research of peripheral adaptations to detraining. Riley and Allen (1973) examined the histochemistry of muscle fiber types following inactivity. Cats received a spinal chord transection which immobilized the tail muscles yet resulted in no denervation. There was no increase in the number of fibers over a 5 month period. The activity of mitochondrial enzymes, represented by NADH diaphorase, had declined noticeably in red fibers by 2 months. All fibers exhibited low diaphorase activity after 5 months.

Immobilization of rat hindlimbs resulted in disuse atrophy in one study (Rifenbrick et al., 1973). Decreases in cytochrome oxidase activity suggested that there were fewer mitochondria in atrophic than in control muscles. The specific activity of malate dehydrogenase in isolated mitochondria was diminished on the first day and decreased to 35% of control by the fifteenth day after immobilization.

Guinea pigs were involved in the original longitudinal study of detraining and muscle histochemistry (Faulkner et al., 1972). Ten animals trained daily for 8 weeks on the treadmill. Measurements were taken 4, 8, and 16 weeks post training to indicate the effect of detraining. There were no significant differences between 4 and 8 weeks of detraining in any of the parameters studied.

Percent red fibers in the plantaris had decreased by 22% after 4 weeks. A concomitant increase of 23% occurred in the white fibers. These changes are indicative of oxidative capacity as SDH activity formed the criteria for designating fiber types. Fiber populations approached control values by 16 weeks of detraining although there was still a slight elevation in the proportion of red fibers. Total fiber concentration was less than the trained value by 15%.

Increases in fiber area were less in trained animals than controls. During the period of detraining growth was again intensified which resulted in larger fiber areas and a heavier total muscle. Houston et al. (1979) reported a similar response in humans. The area of FTa fibers increased significantly with detraining. Increased diffusing distance may result and negatively affect oxidative metabolism.

Muscle enzyme activity in the horse was studied during detraining following a 10 week training program (Guy and Snow, 1977). Six animals completed a program of endurance and sprint training 6 days a week.

Gradual detraining was used in order to avoid fluid accumulation in the legs. Biopsies of 6 muscles were taken 5 and 10 weeks post training. The activity of citrate synthase, representative of citric acid cycle involvement, doubled with training and then significantly decreased in 5 weeks. An insignificant increase occurred during the second 5 weeks of detraining. The activity of the enzyme was still 64% above pretraining values at the conclusion of the detraining period. The authors had no satisfactory explanation for the elevation of activity between weeks 5 and 10 and speculated that an unusual enzyme pattern due to sedentary life may have been responsible. A similar pattern has been discovered in inactive men (Bass et al., 1976).

The effect of decreased work intensity on mitochondrial enzymes was studied by Benzi et al. (1975). Three groups of rats trained 6 times per week for 4 months in programs of graded intensity. Following regular training 2 groups of rats performed the program of next lowest intensity for 2 months. By 40 days the activity levels of SDH had decreased to levels average for the group of preceding intensity.

Detraining has recently been studied in humans. Changes in systemic and local parameters were monitored in previously sedentary men after short term training in 2 studies (Henriksson and Reitman, 1977; Orlander et al., 1977). Thirteen subjects participated in an endurance program for 8 to 10 weeks followed by a 12 week detraining period in the first study. $\dot{V}O_2$ max increased by 19% and the activities of SDH and CYT OX increased by 26% and 35% respectively with training. After 4 weeks of non-training the activity of SDH had decreased significantly and by 6 weeks it had reached control levels. The activity of CYT OX had fallen to pretraining levels by 2 weeks and declined further during the next 4 weeks.

Orlander et al. (1977) trained 16 sedentary men 3 times per week for 7 weeks followed by an 8 week period of inactivity. Training resulted in a significant increase of 6% in $\dot{V}O_2$ max. Intracellular triglyceride content doubled during the program and was restored to pretraining values during the subsequent 8 weeks. Activity of HAD increased by 58% during the first 7 weeks and declined significantly with detraining. Fiber composition was unaffected by training or detraining. Average % ST fibers was 39.7, 35.8, and 36.0 pretraining, post-training and post-detraining respectively.

The effect of 15 days of detraining was studied in 6 well trained runners by Houston et al. (1979). During a peak period of training the athletes were tested for SDH activity, muscle fiber area, and $\dot{V}O_2$ max. These parameters were re-evaluated following the period of inactivity. Maximal aerobic capacity decreased by 4%. The activity of SDH and performance time on a treadmill run to exhaustion were lower by 24% and 25% respectively. Capillary density decreased and fiber composition was unaffected during the period of detraining. A decrease in percent type I fibers from 87% to 57% has recently been reported by an élite athlete after 6 weeks of immobilization (Jansson et al., 1978).

THE TIME COURSE AND RELATIONSHIP OF TRAINING AND DETRAINING

The relationship between the time to adapt with training and detraining is unclear. Many short term studies have found similar elevations and declines in $\dot{V}O_2$ max (Fardy, 1969; Smith and Stransky, 1976) yet others report a significantly longer retention period

(Cureton and Phillips, 1964; Fringer and Stull, 1974). The response of oxidative enzymes has not been as consistant as changes in $\dot{V}O_2$ max. Although Yakovlev (1950) claimed that the enzymes of aerobic metabolism were the first to increase and the last to decrease on breaking training recent studies are in disagreement. Three weeks of training caused an increase in SDH activity of only 11.5% as compared to an increase of 25.6% after 8 weeks, but loss of activity was much more rapid (Henriksson and Reitman, 1977). After 4 weeks of detraining there was a significant decrease and activity of the enzyme had returned to pretraining levels by 6 weeks. Losses of SDH activity of 24% in 15 days and of 50% in 4 to 6 weeks were found by Houston et al. (1979) and Saltin et al. (1977b).

Other enzymes may respond differently to detraining. Orlander et al. (1977) reported an increase of CS activity of 18.4% during 7 weeks of training and an additional increase of 2.2% in an 8 week detraining period. Training had no effect on CYT OX activity in this study although an increase of 34.7% was found by Henriksson and Reitman (1977). These authors reported a decline in CS activity to pretraining levels after 2 weeks of detraining, and further decreases during the next 6 weeks. Intensity of training has been shown to effect enzyme changes (Fitts et al., 1975) but its affect on changes with detraining is yet to be discovered.

Two prevalent theories exist concerning the relationship of systemic and local factors with training and detraining. Saltin et al. (1977b) found no significant differences between relative changes in variables representative of systemic and local response with conditioning programs of 8 to 10 weeks duraction. He concluded that the oxidative potential of muscle may play a crucial role in the determination of $\dot{V}O_2$ max.

Recent studies have stressed the separation of systemic and local training effects (Henriksson and Reitman, 1977; Orlander et al., 1977; Houston et al., 1979; Orlander et al., 1980) arguing that the more rapid decline in oxidative enzymes than $\dot{V}O_2$ max indicates that the oxidative potential of muscle is not a determinant for maximal aerobic power. Although increases of 11% were found in $\dot{V}O_2$ max, SDH activity, and CYT OX activity during the initial 3 weeks of training elevations of 12.6%, 20.5%, and 32.0% had occurred in the above parameters after 5 weeks (Henriksson and Reitman, 1977). Activities of both enzymes had reverted to pretraining values by 6 weeks although 16% of the increased aerobic capacity was retained. Houston et al. (1979) also reported variable changes between central and local factors with detraining. Decreases of 4% and 24% occurred in $\dot{V}O_2$ max and SDH actively during 2 weeks of inactivity by well trained runners.

The time course of changes in fiber subgroups, fiber area, and muscle capillarization is yet to be established. Brown (1976) demonstrated new capillary growth before metabolic adaptation in rabbits. The hypothetical detraining model of Saltin et al. (1977b) however, envisions a much more rapid decline of enzyme activity than of capillarization with detraining. It has been suggested that the sequence of return to pretraining values may progress from changes in area of ST and FTa fibers, decreased oxidative enzyme activity, conversion of FT fiber subgroups, and loss of $\dot{V}O_2$ max to decreases in capillarization over a period of 6 months.

Separation of systemic and local training and detraining effects

TABLE 2.6

TIME COURSE OF CHANGES WITH TRAINING AND DETRAINING (Henriksson and Reitman, 1977)

(% change from pretraining values)

	TRAINING			DETRAINING			
WEEK	3	5	8	2	4	6	12
$\dot{V}O_2$ max	11.1	12.6	18.6	18*	16*	16	
SDH	11.5	20.5	25.6	15*	11*	1	
CYT OX	11.0	32.0	43.7	0			

*approximate values

may reflect the ability to perform at different work intensities. Holloszy (1967) stated that although cardiovascular adaptation may be responsible for increases in maximal aerobic capacity the oxidative capacity of muscle is the limiting factor during prolonged submaximal exercise. Changes in $\dot{V}O_2$ max have been shown to be associated with but not dependent on skeletal muscle oxidative capacity (Henriksson and Reitman, 1977). Decline in performance scores with detraining has been more closely related to local factors of muscle metabolism (Houston et al., 1979) than to changes in $\dot{V}O_2$ max (Ready, 1977) suggesting a relationship between peripheral adaptation and endurance capacity.

Anaerobic threshold (AT) has been proposed as a criterion measure of submaximal fitness (Weltman et al., 1978a). Endurance performance of 22 individuals, grouped by $\dot{V}O_2$ at the onset of metabolic acidosis (AT), led to the conclusion that differences in physiological responses to submaximal work are more directly related to AT than to

$\dot{V}O_2$ max. Rusko et al. (1980) reported significant correlations between AT and oxidative enzyme activities in female cross country skiers. The enzyme activities were not significantly related to $\dot{V}O_2$ max. Muscle respiratory capacity was found to be significantly related to both $\dot{V}O_2$ max and the lactate threshold by Ivy et al. (1980). The porportion of slow twitch muscle fibers also correlated positively with lactate threshold. Other studies have found no significant relationship between fiber type and AT (Rusko et al., 1980).

Davis et al. (1979) reported a significant increase in AT following endurance training in sedentary males. The response of AT to detraining has not yet been established.

SERUM LIPID AND LIPOPROTEIN ADAPTATIONS TO ENDURANCE TRAINING AND DETRAINING

Changes with Training

The focus of research into the effect of exercise on serum lipids has traditionally been directed toward serum cholesterol and triglyceride. Only recently has the effect of training on serum lipoproteins been examined.

Increases in serum cholesterol have been documented following acute exercise (Naughton and Balke, 1964; Jirka and Dolezel, 1968) and are thought to reflect transient mobilization of fuel. Serum triglycerides undergo a similar response (Cohen and Goldberg, 1960; Chinnici and Zauner, 1971) which may be reversed during longer sessions of activity. Carlson and Mossfeldt (1964) found a significant reduction of serum triglyceride in men after 9 hours of skiing.

The response of serum cholesterol to chronic activity has been inconsistent. The majority of studies of healthy subjects have reported decreases following training (Golding, 1961; Shane, 1966; Mann et al., 1969; Wood et al., 1976) while others have found no change (Holloszy et al., 1964; Goode et al., 1966; Milesis, 1974; Lehtonen and Viikari, 1978a). Subsequent studies have revealed that the contradiction results from expression of total serum cholesterol with no indication of its distribution among lipoprotein fractions (Lopez, 1976). The confounding effects of weight loss and food intake on serum cholesterol also make it a poor indicator of the lipid response to exercise (Altekruse and Wilmore, 1973).

Reductions in serum triglyceride following training have been more consistent (Holloszy et al., 1964; Goode et al., 1966; Hunter et al., 1972). This response is felt to reflect the cumulative effect of exercise and to persist for up to 2 days (Oscai et al., 1972). Some studies have reported no change in serum triglyceride with chronic exercise (Hoffman et al., 1967; Milesis, 1974; Lewis et al., 1976). Exercise programs have also been used effectively to normalize serum triglyceride (Oscai et al., 1972; Lampman et al., 1977) and cholesterol (Lampman et al., 1977) in hyperlipidemic patients.

The effect of exercise on different lipid components of the lipoproteins is unequal. Carlson and Mossfeldt (1964) reported a significant reduction of serum triglyceride in healthy persons after 9 hours of exercise. Seventy-five percent of the decrease resulted from a 50% drop in triglyceride content of the very low density lipoproteins (VLDL). Triglyceride also decreased in the low density lipoprotein (LDL) fraction. The cholesterol content was not changed significantly in any of the lipoproteins although the ratio of cholesterol to phospholipid increased in high density lipoproteins (HDL).

The triglyceride content of VLDL also decreased in a group of men who walked 50 km a day for 10 days (Carlson and Frosberg, 1971) and accounted for 66% of the total reduction in serum triglyceride. Smaller decreases occurred in the LDL and HDL. There was no change in the cholesterol content of the lipoproteins.

Changes in the relative proportion of lipoproteins with chronic exercise have been reported, generally decreases of serum VLDL and LDL and an elevation of HDL (Lopez, 1976). In an early study by Hoffman et al. (1967) it was found that air force officers who had engaged in regular activity for 1 year had lower LDL concentrations than sedentary controls. No differences were apparent in the VLDL fraction. Martin et al. (1977) compared the lipid profiles of 28 elite runners to sedentary controls. The athletes had significantly lower LDL-cholesterol and higher HDL-cholesterol fractions than the less active individuals. Two similar studies found the distribution of plasma lipoproteins to vary between runners and controls (Wood et al., 1976, 1977). Lower total plasma cholesterol and LDL-cholesterol and higher HDL-cholesterol were reported for the active group. The ratio of HDL-cholesterol to LDL-cholesterol was also larger for the runners.

Recent studies have been concerned with HDL and physical activity. Trained cross-country skiers were compared to sedentary controls in one investigation (Enger et al., 1977). Significantly higher HDL-cholesterol and HDL-cholesterol to total-cholesterol ratios were reported for the athletes. There was also a significant trend toward progressively higher HDL-cholesterol in the fastest and best trained skiers. Lehtonen and Viikari (1978a) examined the effect of vigorous activity at work on HDL-cholesterol. Lumberjacks, when compared to sedentary electricians, demonstrated significantly higher concentrations. Plasma total cholesterol was higher in the lumberjacks, yet there was no difference in (VLDL and LDL)-cholesterol values.

The response of serum lipoproteins to short term training has not been documented as well as the cross-sectional comparisons between groups. One 10 week program of regular walking and jogging resulted in decreases of LDL and VLDL of 7% and 13% respectively, and an increase in HDL of 19% (Altekruse and Wilmore, 1973). Lopez et al. (1974) found decreased pre-β-lipoproteins (VLDL) and β-lipoproteins (LDL) and elevated α-lipoproteins (HDL) after subjects had participated in a 7 week program of regular activity. Studies by Roundy et al. (1978) and Ratliff et al. (1978) also demonstrated significant shifts from lighter density to heavier density lipoproteins following chronic exercise of 10 and 20 weeks duration.

It has recently been hypothesized that the exercise mediated increase in serum HDL-cholesterol may only result after prolonged training (Hartung and Squires, 1980). Several longitudinal studies of 10 to 12 weeks duration have failed to find significant increases in HDL-cholesterol (Weltman et al., 1978b; Lipson et al., 1979; Squires et al., 1979).

Few studies have related changes in serum lipids and lipoproteins to the intensity or type of exercise. An inverse correlation was reported between the cholesterol content of serum β-lipoproteins and type of physical activity by Todorvic (1971). One investigation found that runners who averaged more than 70 km per week had significantly higher serum HDL-cholesterol concentrations than less active athletes (Lehtonen and Viikari, 1978b). A positive correlation between mileage run and serum HDL-cholesterol has also been reported by Hartung and Squires (1980) for 2 groups of runners.

It is not known conclusively whether exercise affects the synthesis or catabolism of various lipid fractions (Lopez, 1976). Although there is no indication of cholesterol synthesis with exercise it is reported to increase with weight gain (Sodky and Kudchodkar, 1973). Cholesterol catabolism and oxidation increase after exercise (Malinow and Perley, 1969). Synthesis of triglyceride is reduced during exercise (Carlson and Mossfeldt, 1974), and the enzymes of triglyceride metabolism are increased with chronic activity. Lipoprotein lipase (LPL) has been reported to increase in rats (Nikkila et al., 1963; Borenstajn et al., 1975) and man (Nikkila et al., 1978) following training.

Lipoprotein metabolism is also affected by exercise. Reductions in VLDL and LDL and elevations of HDL have consistently been reported with training. The activities of LPL (Nikkila et al., 1978) and lecithin cholesterol acyltransferase (LCAT) (Lopez et al., 1974), enzymes of lipoprotein metabolism, have also been reported to increase with chronic exercise. It has been proposed that reduced triglyceride concentrations in long distance runners as compared to controls may result from increased LPL activity (Nikkila et al., 1978).

Changes with Detraining

Reductions in serum cholesterol which resulted from training did not persist during short periods of detraining in 2 early studies. Rochelle (1961) and Cureton and Phillips (1964) both recorded elevation of cholesterol to pretraining values shortly after the cessation of regular exercise. Rats were trained daily on the treadmill for 8 weeks by Watt et al. (1972). Running duration was progressively increased from 20 to 90 minutes throughout the study. Following 8 weeks of detraining the serum cholesterol and triglyceride levels remained significantly lower than those in untrained control rats.

A study of varsity football players had conflicting results (Penny et al., 1975). Six athletes and 6 sedentary controls were tested at the end of the competitive season and 3, 6, and 9 weeks after its completion. No significant differences in serum cholesterol were reported between the means of the football and control groups on any test. Both groups revealed a decrease in serum cholesterol during the detraining period.

Inconclusive results concerning the response of serum cholesterol to detraining were also reported by Campbell and Lumsden (1967). Subjects participated 3 times a week for 10 weeks in a program of interval running. A 10 week period of inactivity followed. Morphological configuration influenced the results. Slim subjects demonstrated a large increase in serum cholesterol with training and a slight decrease during detraining. Serum cholesterol declined in muscular and obese subjects with training and was elevated significantly during detraining.

An extensive review of the literature failed to reveal any studies which examined the effect of detraining on serum lipoproteins.

CHAPTER III

METHODOLOGY

SUBJECTS

Twenty-six healthy male volunteers between the ages of 18.9 and 31.4 years ($\bar{x} = 25.0$) participated in the study. Three subjects withdrew before completion of the testing sessions and 2 were dropped from the study because of large changes in their activity levels. Participants were requested to maintain their regular exercise pattern and diet throughout the program and both exercise and diet behaviour was monitored on a regular basis.

PROCEDURES

The following dependent variables were measured at 3 week intervals during the study:

- weight (kg)
- total serum cholesterol (mg/100 ml)
- serum HDL-cholesterol (mg/100 ml)
- serum LDL + VLDL-cholesterol (mg/100 ml)
- serum triglyceride
- maximum oxygen consumption ($\ell \cdot min^{-1}$)
- maximum oxygen consumption ($ml \cdot kg \cdot min^{-1}$)
- heart rate at 117.6 watts (BPM)
- heart rate at 176.5 watts (BPM)
- oxygen consumption at 117.6 watts ($\ell \cdot min^{-1}$)
- oxygen consumption at 176.5 watts ($\ell \cdot min^{-1}$)
- maximum ventilation (BTPS) ($\ell \cdot min^{-1}$)

- maximum heart rate (BPM)
- power output at $\dot{V}O_2$ max (watts)
- anaerobic threshold expressed as power output (AT-PO) (watts)
- anaerobic threshold expressed as % $\dot{V}O_2$ max (AT-VO_2)
- anaerobic threshold expressed as $\dot{V}O_2$ (ATml)

Less frequent measures included the assessment of local muscle characteristics, underwater weighing, and diet evaluation to obtain the following variables:

- muscle SDH activity (μmoles x g^{-1} x min^{-1})
- % ST muscle fibers
- % FT muscle fibers
- % FTa muscle fibers
- % FTb muscle fibers
- % body fat
- % carbohydrate intake
- % protein intake
- % fat intake
- caloric intake

Bicycle Ergometer Test

A modification of the step-wise increment bicycle ergometer test developed by Weltman et al. (1978a) was used to measure maximum and submaximum metabolic variables. An initial load of 90 watts was set and a pedalling rate of 60 RPM was adopted. Subjects were paced by an auditory-visual metronome and a micro-switch counter was used to monitor work output. Heart rate was continuously recorded from a bi-polar chest lead.

Variables dependant upon expired gas analysis were measured by a Beckman Metabolic Measurement Cart. This system contains an OM-11 oxygen analyzer and a LB-2 carbon dioxide analyzer, as well as volume, temperature, and pressure transducers. Data are multiplexed from these sensors and transducers into a memory system and transferred into a calculator. Thirty second recordings were made of expired volume ($\dot{V}_e$, BTPS, $\ell.\text{min}^{-1}$), fraction of expired O_2 and CO_2 (FEO_2, $FECO_2$), respiratory exchange ratio (R), and oxygen consumption ($\dot{V}O_2$) in milliliters per kilogram per minute (ml.kg.min^{-1}) and in liters per minute ($\ell.\text{min}^{-1}$). These calculations are displayed in Appendix A.

Calibration of the metabolic cart was performed prior to and after each test.

Anaerobic Threshold

Estimation of anaerobic threshold from gas exchange variables has been found to be valid and reliable (Davis et al., 1976).

The initial load of 90 watts was increased by 30 watts at 2 minute intervals throughout the test. Values for FEO_2, $FECO_2$, $\dot{V}E$, and R were displayed by the Beckman Metabolic Measurement Cart every 30 seconds. Heart rate was continuously recorded from a bi-polar chest lead and a micro-switch counter was used to monitor work output.

Steady state values for each of the gas exchange variables were plotted against power output at each work level. The criterion for determination of the power output associated with metabolic acidosis was the point of departure from linearity in the $\dot{V}E/\dot{V}O_2$ versus power output curve as indicated by the largest change in the differential of the slope. Similar changes in the $\dot{V}E$, FEO_2, and R versus power output curves were used to assist in the calculation of anaerobic threshold in the few cases where an abrupt change in $\dot{V}E/\dot{V}O_2$ versus power output was not apparent. Appendix B contains a sample of the graphs used to determine threshold for one subject.

Maximum Oxygen Consumption

In order to measure peak $\dot{V}O_2$ on the bicycle ergometer resistance was increased by 30 watts at 1.5 or 2 minute intervals following the determination of anaerobic threshold. Subjects were verbally encouraged to continue until exhaustion or until oxygen consumption levelled off or decreased (within 100 ml) with an increase in workload.

Muscle Biopsy

Muscle biopsies were taken from the vastus lateralis by the method of Bergstrom (1962) in order to determine percent fiber population and succinate dehydrogenase (SDH) activity. The biopsy site was on the lateral side of the thigh, midway between the spina ilica anterior superior and the upper border of the patella, an area of minimal risk due to scarcity of blood vessels and nerves. The operating area was kept sterile and the biopsies were performed by a physician.

Incisions were made on the right thigh and one muscle core removed. Tissue was divided in half and one sample was frozen within five seconds in isopentane cooled in liquid nitrogen for biochemical determinations. Fat and connective tissue were dissected from the other section of tissue core, which was then mounted in OCT mounting medium on a cork and frozen in isopentane cooled in liquid nitrogen for histochemical determinations. Both samples were stored at -60°C until analyzed.

Fiber types were identified on the basis of the myofibrillar ATPase reaction (Padykula and Herman, 1955) following preincubation in acetate buffer at pH 4.3 or pH 4.61 or in glycine buffer at pH 10.3 (Houston, 1978) (Appendix C). Type I, IIa and IIb fibers were distinguished for most samples. The muscle tissue was also stained for NADH-diaphorase activity to indicate oxidative capacity (Appendix D). Serial sections, 10 um thick, were cut in a cryostat at -20° centigrade, picked up onto a cover slip and dried at room temperature for 24 hours before being stained. Fiber types were counted from photomicrographs and an average of 264 fully intact fibers were used to calculate the fiber type percent for each sample.

The activity of SDH was measured by the fluorometric technique of Lowry and Passonreau (1972). Fluorometry is a method of measuring the fluorescence, or instantaneous emission of light, from a molecule or atom which has absorbed light. The rate of change of fluorescence with time, Δ F/minute, is directly proportional to the concentration of the enzyme being measured provided the concentrations of substrates and auxillary enzymes are in excess allowing the enzyme under study to be the rate limiting step in the reaction. All reactions are NADH or NADPH coupled to provide a molecule with measurable fluorescence. Fluorometry is advantageous in that it is precise enough to accurately measure enzyme activities of muscle samples as small as one milligram wet weight.

Frozen muscle samples were thawed in ice-cold 0.1 M Tris buffer (pH 7.5) and blotted to remove any blood. Connective tissue was removed and each sample was weighed to the nearest one-tenth of a milligram on a Mettler H20T analytical balance. A Potter-Elvehjem glass homogenizer was used to homogenize samples five times for three seconds each in 0.5 ml of ice-cold 0.1 M Tris buffer at pH 7.5. To prevent denaturation of enzymes from heat build up each grinding was separated by 30 seconds. The homogenizers were placed in ice-cold water baths to minimize the heat. Samples were poured off and homogenizers washed with an additional 2.5 ml of buffer to give a final dilution of 3 ml per sample. (See Appendix E for homogenization procedure). Notable pieces of connective tissue were removed and weighed. Subtraction of this weight from the weight of the original sample gave a more accurate wet weight of muscle tissue.

The activity of SDH was determined from the whole muscle homogenate (the procedure is reported in Appendix F). Activity was expressed in μmoles per gram wet weight per minute (μmoles x g^{-1} x min^{-1}). Samples were held in matched 3 ml culture tubes, and primary and secondary filters with excitation wavelengths of 364 and 465 nanometers respectively were used in a Turner model III flurometer. Blank samples containing everything but the fluorescent substances were also recorded. Standards for NADH were measured on the fluorometer to give the value in μmoles per milliliter for a change of one unit in fluorescence. The assay was determined at 21° centrigrade using the Turner temperature regulatory door and a Thelco water bath.

Body Composition, Diet, and Activity Record

Percent body fat was estimated by the underwater weighing technique described by Sloan (1962). A modification of the body density formula derived by Brozek et al. (1973) was used for the above measurement. See Appendix G for a sample calculation.

Subjects were asked to submit periodic dietary and activity records. Computerized analysis of the diet determined caloric intake and nutrient content. See Appendix H for a sample printout.

Quantification of activity during random periods of time was done using the Activity Assessment Form in Appendix I.

Blood Analysis

Blood samples were taken, after a 14 hour fast and a 72 hour alcohol restriction, from the antecubetal vein for determination of serum cholesterol, triglyceride, HDL-cholesterol, and (VLDL + LDL)-cholesterol. All samples were taken in the morning and subjects were requested to refrain from exercise for 14 hours prior to the blood drawing.

Seven milliliters of blood were taken from each subject. The samples were spun in a centrifuge at 3,000 g's for 10 minutes. Five hundred ml of serum were transferred to a small glass test tube (100 x 12 mm) using a volumetric pipette. See Appendix J for the procedure required to fractionate HDL and (LDL + VLDL) cholesterol.

Concentrations of the serum lipids and lipoproteins were determined by use of prepared kits (Boehringer Mannheim Diagnostica). Serum triglyceride and cholesterol were measured by modifications of the procedures described by Bucolo et al. (1973) and Klose et al. (1975), and Allain et al. (1974) respectively. The concentration of serum HDL-cholesterol was determined by the procedure of Burstein et al. (1970) and Bagdade et al. (1977). These procedures are described in Appendix K. (VLDL + LDL)-cholesterol was estimated from the difference between total serum cholesterol and HDL-cholesterol. All assays were done in triplicate and instruments were regularly calibrated in the recommended fashion.

EXPERIMENTAL DESIGN

Prior to the start of the treatment period all 26 subjects were tested on all measures except muscle biopsies and randomly assigned to either the experimental group (E, n = 14) or the control group (C, n = 12). Predicted $\dot{V}O_2$ max was then used as the criterion for designating one-half of each group (E and C) as either high fit or low fit.

Six subjects from both the experimental and control groups were randomly chosen to receive muscle biopsies at the commencement of the study. The remaining 14 participants were tested on this parameter following the training period. Biopsies were again taken from 6 members of each group following completion of the detraining process.

The total duration of the experiment was 18 weeks. After administration of the pre-tests the experimental group participated in 9 weeks of training followed by 9 weeks of detraining. Metabolic measurements, including the tests for $\dot{V}O_2$ and AT, as well as lipid analysis, were done at 3 week intervals during the study.

All subjects received one muscle biopsy at either week 0 or week 9 of the training program. Only 12 of 21 remaining subjects were available for second biopsies at the end of 18 weeks; 3 declined the test and 6 were unavailable. Body composition was evaluated at 3 points during the study: before and after the training program and following detraining. Diet and activity analysis was done during both the training and detraining periods.

The experimental design is outlined below:

		TRAINING							DETRAINING		
WEEK		0		3		6		9	3	6	9
GROUP	E	0_{11}	X	0_1	X	0_1	X	0_{11}	0_1	0_1	0_{11}
	C	0_{11}		0_1		0_1		0_{11}	0_1	0_1	0_{11}

X = Training stimulus
0_1 = Metabolic and blood measures
0_{11} = All measures

STATISTICAL ANALYSIS

A three way ANOVA with repeated measures (Kirk, 1968) was used to determine the significance of differences between and within groups for the following dependant variables: anaerobic threshold, maximum and submaximum oxygen intake and heart rate, maximum ventilation, power output at $\dot{V}O_2$ max, serum lipids and lipoproteins, diet characteristics, and per cent body fat. To ascertain whether significant differences existed between groups in muscle fiber types and SDH activity a two way ANOVA was used.

Prior to the analysis a 0.05 level of statistical significance was established. When a significant F was obtained a post hoc comparison of means was made using a Scheffé test (Kirk, 1968). Planned comparisons, between the exercise and control groups and between tests for the exercise group, enabled further investigation of the results.

TRAINING PROGRAM

Members of the experimental group participated in a continuous training program on the bicycle ergometer 4 times a week for 9 weeks. Training intensity was at 80% $\dot{V}O_2$ max and each session lasted for 30 minutes. Similar training programs have demonstrated significant improvements in fitness (Henriksson and Reitman, 1977). Heart rates were continuously monitored with a cardiotachometer during each exercise bout.

During weeks 3 and 6 of the training program subjects were required to perform a bicycle ergometer test instead of 2 of their training sessions. Calculation of $\dot{V}O_2$ max at these points enabled re-establishment of the training program at an intensity equal to 80% of the subject's maximum.

CHAPTER IV

RESULTS AND DISCUSSION

The results and discussion are presented in seven sections: physical characteristics, body composition and diet analysis, the intensity of training, and the responses to training and detraining of metabolic parameters, anaerobic threshold, local muscle, and serum lipids and lipoproteins. A general discussion integrating adaptations with training and detraining concludes the chapter.

Summary tables and graphical representations of the results are presented in this chapter. The original data and statistical analyses are located in the Appendices. Statistical significance was established as $p \leq 0.05$. Planned comparisons between groups, and between tests for the exercise group, were performed when F values approached significance.

PHYSICAL CHARACTERISTICS

The height, weight and age of the subjects at the time of the pre test are presented in Table 4.1. There were no significant differences in weight between or within groups at the pre test or during the study.

BODY COMPOSITION AND DIET ANALYSIS

The analysis of variance indicated no significant differences between or within groups for percent body fat; simple main effects tests revealed a significant difference between weeks 1 and 4 for the exercise group (F = 1, (32) = 20.58). Decreases in body fat have been

TABLE 4.1

PHYSICAL CHARACTERISTICS OF THE SUBJECTS ($\bar{X} \pm SD$)

GROUP	HEIGHT (cm)	WEIGHT (kg)	AGE (yrs)
Exercise (n = 12)	179.8 ± 4.5	76.7 ± 12.2	25.0 ± 3.6
Control (n = 9)	180.3 ± 5.7	73.7 ± 9.7	25.0 ± 3.2

reported previously for trained males (Wood et al., 1976). Seasonal variation in body composition may have accounted for the decline in both groups following detraining. Values for mean percent body fat appear in Table 4.2.

TABLE 4.2

PERCENT BODY FAT BEFORE AND AFTER TRAINING AND DETRAINING ($\bar{X} \pm SD$)

GROUP	PRE TEST	POST TEST	POST DETRAINING
Exercise (n = 12)	17.8 ± 6.0	15.0 ± 4.5	13.4 ± 4.7
Control (n = 8)	12.8 ± 3.7	12.4 ± 5.0	9.6 ± 3.8

Evaluation of diet on 2 occasions during the study showed no significant differences between or within groups for caloric intake, percent protein, percent carbohydrate or percent fat (Table 4.3).

It has been suggested that the changes in diet and weight which often occur during an exercise program may affect the response of serum lipids during training (Alterkruse and Wilmore, 1973). The absence of significant differences in weight, caloric intake, and diet composition during this study allow more rigorous conclusions to

TABLE 4.3

DIET ASSESSMENT DURING TRAINING (T) AND DETRAINING (D) ($\bar{X} \pm SD$)

GROUP	DAILY CALORIC INTAKE		% PROTEIN		% CARBOHYDRATE		% FAT	
	T	D	T	D	T	D	T	D
Exercise **(n = 11)**	2590.4 ± 655.4	3052.2 ± 1242.5	17.8 ± 2.4	15.2 ± 1.7	43.8 ± 7.7	42.4 ± 6.9	38.7 ± 6.8	40.3 ± 6.3
Control **(n = 6)**	3119.3 ± 950.3	2767.6 ± 739.9	16.8 ± 2.7	16.1 ± 1.4	50.8 ± 4.4	46.8 ± 3.6	34.2 ± 4.9	35.2 ± 4.4

be made concerning the effect of training and detraining on serum lipids and lipoproteins. Campbell and Lumsden (1967) reported a significant interaction between body composition and changes in serum cholesterol with training and detraining. The decrease in percent body fat in the experimental group in the present investigation may have had an effect on the lipid response to exercise.

THE INTENSITY OF TRAINING

Subjects trained at the heart rate required to maintain a power output equivalent to 80% of their $\dot{V}O_2$ max. Training workloads and target heart rates were re-evaluated at 3 week intervals during the study. The average power output increased by 34% after 9 weeks of training. A record of work completed by each subject during the program appears in Appendix M.

RESPONSE OF METABOLIC PARAMETERS TO TRAINING AND DETRAINING

Maximum Work

Significant group x time interactions occurred in $\dot{V}O_2$ max expressed in $\ell . \text{min}^{-1}$ (F = 1, (84) = 2.77, $p < 0.016$) and in ml.kg.min^{-1} (F = 6, (84) = 3.395, $p < 0.005$). Tests for simple main effects indicated significant differences between the pre test and all other tests, and between test 4 and tests 2, 3, and 7 in relative $\dot{V}O_{2\ \text{max}}$ (ml.kg.min^{-1}) for the experimental group. Absolute $\dot{V}O_2$ max ($\ell \cdot \text{min}^{-1}$) differed significantly between test, and all other tests, and test 4 and tests 3, 6, and 7. These findings are presented in Table 4.4.

The pretraining values for $\dot{V}O_2$ max are similar to those previously reported for young, sedentary males (Ekblom, 1969). Increases of

TABLE 4.4

MEASURES OF THE METABOLIC RESPONSE TO TRAINING AND DETRAINING ($\bar{X} \pm SD$)

TEST	EXERCISE GROUP 1	2	3	4	5	6	7
$\dot{V}O_2$ max (ml.kg.min^{-1})	45.7 ± 8.4	56.8 ± 9.1	56.0 ± 8.2	64.2 ± 9.5	59.3 ± 6.4	57.5 ± 9.7	57.3 ± 8.8
$\dot{V}O_2$ max (ℓ.min^{-1})	3.46 ± .7	4.27 ± .6	4.17 ± .4	4.73 ± .7	4.39 ± .5	4.21 ± .7	4.21 ± .5
SUBMAX HR (BPM)	132.4 ± 14.0	120.7 ± 12.9	123.8 ± 11.5	118.1 ± 13.8	120.2 ± 11.4	124.0 ± 9.8	123.1 ± 12.9
				CONTROL GROUP			
$\dot{V}O_2$ max (ml.kg.min^{-1})	45.4 ± 5.5	52.5 ± 4.7	50.5 ± 7.0	51.4 ± 5.1	51.0 ± 7.1	51.0 ± 5.7	53.8 ± 7.1
$\dot{V}O_2$ max (ℓ.min^{-1})	3.32 ± .6	3.90 ± .5	3.73 ± .5	3.78 ± .4	3.70 ± .3	3.70 ± .4	3.89 ± .4
SUBMAX HR (BPM)	128.1 ± 13.4	134.6 ± 14.5	134.7 ± 7.7	131.9 ± 10.9	131.6 ± 16.0	131.0 ± 14.5	129.3 ± 17.5

24.3% and 15.6% in the exercise and control groups between test 1 and test 2 suggest that the initial response was a peak value for the bicycle ergometer and may have been limited by local fatigue. Measures of predicted $\dot{V}O_2$ max, used to designate subjects as 'hi fit' or 'lo fit', also demonstrate that the initial value for maximal aerobic capacity may have been low (Appendix N).

Although the training program resulted in an average increase of 40.5% in $\dot{V}O_2$ max for the exercise group values were also elevated by 13.2% in the control subjects after 9 weeks. Approximately 28% of the difference in the exercise group was not accounted for by the change in control group values. Similar changes in response to short term exercise programs have been reported in the literature (Saltin et al., 1977a).

Increases in $\dot{V}O_2$ max took place during the initial and final 3 weeks of training. The plateau between weeks 3 and 6 may have resulted from under prescription of exercise intensity. It is also possible that the division of training effects into 2 stages may be due to differential changes in various underlying mechanisms contributing to $\dot{V}O_2$ max.

The greatest decline in maximal aerobic power occurred during the first 3 weeks of detraining with a loss of 7.6%. Decreases of 3.0% and 0.5% occurred during 2 subsequent 3 week periods. The significant difference between test 1 and test 7 indicates that some training gains were retained following the 9 week detraining period.

Fitness level has been found to affect the response to endurance training (Saltin, 1969; Knuttgen et al., 1973). Changes in $\dot{V}O_2$ max in the present study did not differ significantly between subjects classified as 'hi fit' or 'lo fit'. Possible explanations for this discrepancy include the classification criterion (predicted $\dot{V}O_2$ max), and prescription of relative training intensities. Maximal heart rate has been reported to decrease slightly (Saltin, 1969) or to remain unchanged (Ekblom, 1969) in response to regular exercise. The literature supports the lack of significant change found in the present study. Maximal ventilation also remained unchanged in response to training and detraining.

Submaximal Work

A significant group x time interaction was obtained for steady state heart rate at 117.6 watts ($F = 6, (84) = 3.905, p < 0.002$). Post hoc analysis revealed that differences between test 1 and tests 4 and 5 in the training group were responsible for the significance (Table 4.4). No significant interactions occurred for steady state heart rate at 176.5 watts. Fitness level significantly affected both submaximal heart rates. The main effects obtained were $F = 1, (14) = 10.696, p < 0.006$, and $F = 1, (14) = 6.477, p < 0.0023$ at 117.6 at 176.5 watts respectively.

Decreases in submaximal heart rate with training are well documented (Frick et al., 1967; Saltin et al., 1969). Although steady state heart rate at 117.6 watts declined by 8.7% in response to the initial 3 weeks of training it was 9 weeks before a significant training effect occurred. Following 3 weeks of detraining heart rate was still significantly lower than the initial value. Six and 9 weeks post training there were no significant differences from the pre test heart rate although the average value remained 6.9% lower.

Several investigators have reported significant increases in submaximal heart rate in response to similar periods of non-training (Michael and Gallon, 1959; Hammer, 1965; Michael et al., 1972).

Steady state oxygen consumption at 117.6 and 176.5 watts was not significantly affected by the training program or fitness level of the subjects. Mechanical efficiency, as measured by oxygen consumption, has been reported to remain unchanged (Holloszy, 1973) and increase as the result of training (Ekblom, 1969).

RESPONSE OF ANAEROBIC THRESHOLD TO TRAINING AND DETRAINING

Significant interactions were obtained for anaerobic threshold, expressed as ml oxygen consumption per kg body weight, group x time $F = 6, (84) = 4.65, p < 0.001$, and expressed as power output (watts), group x time $F = 6, (84) = 5.938, p < 0.05$. There were no significant interactions when threshold was expressed as a percent of $\dot{V}O_2$ max.

Comparison of means for the exercise group revealed significant differences in ATml between test 1 and all other tests and test 4 and tests 2, 6, and 7. When AT was expressed relative to power output (AT-PO) significance was found between test 1 and tests 3 and 4, test 3 and tests 2, 5, 6, and 7, and test 4 and tests 2, 5, 6, and 7. Mean values of AT during training and detraining are located in Table 4.5

Little has been reported in the literature regarding the response of AT to endurance training. Davis et al. (1979) reported increases of 44% and 15% in AT (ℓmin^{-1}) and AT-$\dot{V}O_2$ following a 9 week exercise program. Work intensity was 50% between $\dot{V}O_2$ max and AT. The training intensity of 80% $\dot{V}O_2$ max employed in the present investigation was also

TABLE 4.5

MEASURES OF THE RESPONSE OF ANAEROBIC THRESHOLD TO TRAINING AND DETRAINING ($\overline{X} \pm SD$)

TEST	1	2	3	4	5	6	7
				EXERCISE GROUP			
AT-$\dot{V}O_2$	64.9 ± 14.4	68.8 ± 10.2	77.9 ± 6.7	77.5 ± 11.0	70.4 ± 11.7	70.4 ± 13.6	69.3 ± 13.1
AT ml	29.2 ± 5.9	38.9 ± 7.4	43.6 ± 7.3	49.0 ± 5.0	42.0 ± 9.6	40.5 ± 9.8	39.8 ± 9.6
AT-PO (watts)	197.9 ± 26.6	216.6 ± 35.5	248.7 ± 27.5	264.7 ± 26.3	227.3 ± 41.8	216.6 ± 35.5	216.6 ± 30.2
				CONTROL GROUP			
AT-$\dot{V}O_2$	79.9 ± 12.9	75.5 ± 9.9	74.1 ± 11.1	76.5 ± 11.7	82.1 ± 7.1	80.2 ± 11.8	74.5 ± 9.3
AT ml	36.7 ± 9.5	39.8 ± 7.3	37.4 ± 7.3	39.6 ± 8.3	41.6 ± 4.8	40.9 ± 8.1	40.1 ± 7.3
AT-PO (watts)	214.3 ± 40.6	210.1 ± 39.5	205.9 ± 24.0	214.3 ± 40.6	218.5 ± 33.3	210.1 ± 39.5	218.5 ± 41.0

above the mean AT-$\dot{V}O_2$ max.

An increase in ATml of 67.8% resulted from the 9 week program. The largest elevation occurred in the initial 3 weeks. The 12% improvements between weeks 3 and 6, and 6 and 9 were not statistically significant, although test 4 was significantly different from test 2.

The loss of training gains in ATml was significant following 6 and 9 weeks of detraining. Although a rapid decrease of 14.3% took place during the initial 3 weeks it was not significant. When expressed relative to power output (AT-PO) however, the initial decline becomes significant.

AT-PO and AT-$\dot{V}O_2$ increased by 33.8% and 19.4% during the training program. The largest improvements occurred between weeks 3 and 6. Following the detraining period ATml, AT-PO, and AT-$\dot{V}O_2$ remained elevated by 36.3%, 9.4%, and 6.8% above their initial mean values respectively. Anaerobic threshold did not change significantly in the control group.

RESPONSE OF LOCAL MUSCLE TO TRAINING AND DETRAINING

Fiber Distribution

Muscle biopsies were taken from the vastus lateralis on 3 occasions: pre test, post test and post detraining. Each subject had a maximum of 2 biopsies: an assumption of homogenity in fiber types among groups was made in order that the samples could be statistically analyzed. Distributions of approximately 50:50 for ST and FT fibers have frequently been documented in untrained males (Gollnick et al., 1972; Costill et al., 1976a).

Classification of fibers as ST, FTa, and FTb after staining for

ATPase (Houston et al., 1979) was possible for 8 samples from the exercise group and 7 from the control group. An average of 264 fibers were counted in each section. Two way analysis of variance, and analysis with number of fibers as a covariate, resulted in no significant differences within or between groups for percent distribution of ST, FTa or FTb fibers.

Slow twitch fibers averaged 55.8 and 58.9 percent respectively for the exercise and control groups. The effect of endurance training on the subgroup population of FT fibers may not be apparent due to the small cell sizes. Several studies have reported a shift from FTb to FTa following aerobic activity (Green et al., 1979; Andersen and Henriksson, 1977a; Jansson and Kaijser, 1977). A similar trend was found in the present investigation with an average FTa distribution for 7 subjects of 35.3% prior to training and 38.7% following the 9 week program. An inverse change occurred in the FTb population: from 10.1% to 6.3 %. These alterations were reversed during the detraining period.

SDH Activity

SDH activity in the vastus lateralis was also determined pre and post training and post detraining. Values were not determined for each subject every time, however, and conclusions are limited in that group means were analyzed statistically.

Mean activity averaged 4.63 and 4.58 μmoles x g x min^{-1} for 4 members each of the exercise and control group at the pre test. This value is similar (Gollnick et al., 1972) or slightly lower (Costill et al., 1976a; Jansson and Kiajser, 1977) than those previously reported for untrained males. Variations in measurement technique may account for this slight discrepancy. Several studies have documented elevated activity of SDH in endurance athletes. Activities as high as 14.7 (Houston et al., 1979) and 16.6 (Costill et al., 1976a) μmoles x g x min^{-1} have been found in the vastus lateralis of distance runners. Changes in enzyme activity are specific to the muscle groups employed in training (Benzi et al., 1975) making the vastus lateralis an ideal biopsy site for subjects trained on the bicycle ergometer.

Although there were no significant differences between groups or times in SDH in the present study post test activity was elevated by 42% over the pre test value in the exercise group. The control group varied by only 8% on these 2 measures. SDH activity had returned to the pre training level following 9 weeks of detraining. The absence of sequential values for each subject limits the interpretation of these results. It is apparent, however, that a trend toward increased and decreased activity during short term training and detraining does exist.

TABLE 4.6

SDH ACTIVITY DURING TRAINING AND DETRAINING
(μmoles x g^{-1} x min^{-1}) ($\bar{x}$ ± SD)

GROUP	PRE TRAINING	POST TRAINING	POST DETRAINING
Exercise	4.63 ± 2.40 (n = 4)	6.58 ± 1.33 (n = 6)	4.24 ± 3.11 (n = 6)
Control	4.58 ± 2.99 (n = 4)	4.95 ± 1.30 (n = 3)	3.25 ± 1.66 (n = 6)

RESPONSE OF SERUM LIPIDS TO TRAINING AND DETRAINING

The analysis of variance resulted in no significant interactions for any of the lipid variables. Initial mean values reported in this study are similar to those documented in the literature for serum cholesterol (Lehtonen and Viikari, 1978b; Enger et al., 1977), triglyceride (Holloszy et al., 1964), HDL-cholesterol (Hartung and Squires, 1980) and (VLDL + LDL)-cholesterol (Wood et al., 1976). The reliability of the method used in the measurement of HDL-cholesterol was determined by regular analysis of a known reference sample (Appendix L).

An insignificant decline of serum cholesterol by 3.8% occurred in the experimental group between weeks 3 and 6 of training. This change was reversed during the detraining period. Rochelle (1961) and Cureton and Phillips (1964) have also reported elevation of cholesterol to pre training values shortly after the cessation of training.

Inadequate control over the effect of body weight loss and food intake during many studies (Lopez, 1976) has been in part responsible for changes found in serum cholesterol with training (Mann et al., 1969; Wood et al., 1976). Diet composition, caloric intake, and body weight did not change during the present investigation and therefore did not likely affect the lipid response. Several other studies have reported changes in serum cholesterol following training (Holloszy et al., 1964; Milesis, 1974; Lehtonen and Viikari, 1978a). The lack of consistant findings may be due to variations in the training stimulus, control over extraneous variables, or the differential effect of exercise on the lipoprotein fractions of cholesterol (Lopez, 1976).

Serum triglyceride decreased by 48.8% in the exercise group during the 9 week training program. This change was not statistically significant however, possibly as the result of high intra and inter subject variability. Although most investigations have found decreases in serum triglyceride following chronic exercise (Holloszy et al., 1964; Goode et al., 1966; Hunter et al., 1972) others have reported no change (Hoffman et al., 1967; Milesis, 1974; Lewis et al., 1976). Triglyceride concentration remained elevated above the pre test value by 18.4% after the 9 week detraining period. Watt et al. (1972) found similar retention of training decreases in serum triglyceride and cholesterol following 8 weeks of endurance training and detraining.

The rate of catabolism of circulating triglyceride is determined by the activity of lipoprotein-lipase (LPL). Increases in the activity of this enzyme have been found to occur with training (Borensztajn et al., 1975; Nikkila et al., 1963; Nikkila et al., 1978) and provide a possible explanation for the reduction in serum triglyceride. A change in insulin sensitivity of muscle and adipose tissue (Nikkila et al., 1978), and the elevated release of catecholamines (Golding, 1961) with chronic exercise have been suggested as possible mechanisms by which the enzyme activity is altered.

High inter and intra subject variability in lipoprotein measures may have precluded the discovery of significant changes during this investigation. A definite trend toward elevated HDL-cholesterol was apparent in the exercise group: values remained 10% higher than at the pre test after 3 and 6 weeks of detraining. Four percent of the training gain was maintained after 9 weeks indicating that the mechanisms responsible for the change may be affected gradually.

A slight decrease of 3.5% occurred in mean (VLDL + LDL)-cholesterol in the exercise group during the 9 week program. Values had returned to the initial level by 3 weeks. The discrepancy in the time course of changes in (VLDL + LDL)-cholesterol and HDL-cholesterol give support to a precursor-product theory of lipoprotein metabolisms.

The elevated HDL-cholesterol levels often present in endurance trained individuals (Enger et al., 1977; Wood et al., 1977; Martin et al., 1977) may result from alterations in enzyme activity. A significant correlation of +.72 was found between HDL-cholesterol and LPL activity of adipose tissue in male runners and controls (Nikkila et al., 1978). It is theorized that a precursor-product relationship may exist between VLDL-cholesterol and HDL-cholesterol (Lopez, 1976). In the presence of lecithin:cholesterol acyltransferase (LCAT) and LPL there may be a transfer of triglycerides from VLDL to HDL in exchange for esterified cholesterol. Decreased (VLDL + LDL)-cholesterol and increased HDL-cholesterol found in trained subjects support this theory (Hoffman et al., 1967; Altekruse and Wilmore, 1973; Wood et al., 1977), as does the elevated activity of LCAT) (Lopez et al., 1974). It has also been suggested that the release of newly formed HDL-cholesterol may increase following chronic exercise (Lopez, 1976).

Nine weeks of training may not be sufficient duration to affect lipoprotein metabolism. Most studies report decreased (VLDL + LDL)-cholesterol and increased HDL-cholesterol after long term training (Enger et al., 1977; Wood et al., 1977; Martin et al., 1977). Hartung and Squires (1980) hypothesized that the exercise mediated increase in HDL-cholesterol may take months or years to manifest itself. The failure of several short term exercise programs to result in significant changes in serum lipoproteins is evidence that this may be the case (Weltman et al., 1978b; Lipson et al., 1979; Squires et al., 1979).

The intensity of training may also affect the response of serum lipoproteins. Significant correlations have been reported between mileage and HDL-cholesterol in runners (Lehtonen and Kiikari, 1978b; Hartung and Squires, 1980). The training load of 80% of $\dot{V}O_2$ max employed in the present study was well above the intensity of most running programs, suggesting that duration may have been the factor limiting changes with training. Differential results between the subjects in Hartung and Squire's study (1980) led to the contention that genetic disposition may also affect the response of serum lipoproteins to training.

GENERAL DISCUSSION

The response of several systemic and local muscle parameters to endurance training and detraining has been examined in this investigation. Distinctions between central circulatory changes and changes in local tissue following chronic exercise enable the identification of physiological mechanisms responsible for training adaptations. Greater understanding of the factors contributing to performance, and of optimal training techniques, may result from classification of these mechanisms.

Observation of the time course of adaptation to chronic activity and inactivity is one method by which the differential responses of various physiological parameters may be recognized. It has been suggested that initial increases in $\dot{V}O_2$ max with training may be the result of adaptations in $\dot{Q}$ and SV, and that local changes occur only after a longer period of time (Cunningham and Hill, 1975). Cunningham et al. (1979) reported elevations of $\dot{V}O_2$ max by 22% after 12 weeks of interval or continuous training. Changes in the initial 4 weeks resulted primarily from altered $\dot{Q}$ and SV, measured at 85% of $\dot{V}O_2$ max. $A\text{-}\dot{V}O_2\Delta$ became a significant factor in the increase after the eighth week. Orlander et al. (1977) found changes in oxidative enzyme activities also require significant training durations. No change in cytochrome oxidase activity was reported in men after 7 weeks of training, although an additional 7 weeks resulted in significant increases.

The time course of changes in local muscle and systemic parameters during this investigation are summarized in Table 4.7. The large increase in $\dot{V}O_2$ max during the first 3 weeks of training may partly reflect familiarization to test protocol. Loss of training gains was greatest 3 weeks following the post test; 25% of the increase was retained at weeks 6 and 9.

The response of ATml to the training program was greater than that of $\dot{V}O_2$ max. Nine weeks of training resulted in an increase of close to 70%. One half of the change occurred in the initial 3 weeks of the program. Atml decreased most significantly during the initial detraining period; 36% of the gain was retained at the conclusion of 9 weeks of non-training.

Steady state heart rate for a submaximal load (117.6 watts) was not affected as quickly during the detraining period. Only 2% of the 10% decrease with training was lost after 3 weeks. Nine weeks was insufficient time for a return to pre training baseline values. The activity of SDH was the only parameter to return to its original value

TABLE 4.7

CHANGES IN SYSTEMIC AND LOCAL MUSCLE PARAMETERS WITH TRAINING AND DETRAINING

(Per cent change from pre test value)

	Training			Detraining		
	3 weeks	6 weeks	9 weeks	3 weeks	6 weeks	9 weeks
$\dot{V}O_2$ max ($ml.kg.min^{-1}$)	+24.3	+22.5	+40.5	+29.8	+25.8	+25.4
HR 117.6 (BPM)	-8.7	-6.4	-10.7	-9.1	-6.2	-6.9
ATml	+33.2	+49.3	+67.8	+43.8	+38.7	+36.3
SDH activity* (μmoles x g x min^{-1})	-	-	+43.7	_	_	-7.2

* representative values from different individuals and not pre-post values for the same subject

following the detraining period.

Variations in the time course of training responses may be related to differential changes in central and local parameters. Saltin et al. (1977b) have proposed a theoretical model of the chronological response to exercise. Short term training is believed to enhance the circulatory capacity and ability to utilize oxygen, whereas training of longer than 6 months duration may affect prolonged exercise performance. The increased capacity for submaximal work may result from changes in lactate production and accumulation and from a glycogen sparing effect (Holloszy, 1967). Results of the present study suggest that intensity of training may be the more critical factor in determination of these differential training effects.

Cunningham et al. (1979), in a comparison of interval and continuous training, also found training adaptations to be very specific to exercise intensity.

Several research models have been utilized in the attempt to distinguish between central and local limitations to exercise. Training programs which involve various amounts of muscle mass, and artificial changes in the arterial oxygen content have been used in order to determine whether the limitation to $\dot{V}O_2$ max is central or local (Clausen, 1977; Saltin et al., 1977a). Time course studies also allow investigation of this problem through the identification of those factors which respond in similar manner to a training stimulus.

Parallel adaptation in local muscle and central circulatory parameters would indicate close functional association. Saltin et al. (1977b) reported a close relationship between local and cardiovascular changes during training, and suggested that a peripheral control system may be responsible for changes in submaximal heart rate. He also indicated that changes in $\dot{V}O_2$ max, oxidative enzymes, capillarization and fiber size and composition may occur at similar rates during the initial months of an exercise program.

Recent investigators have found that changes in $\dot{V}O_2$ max and the metabolic potential of muscle are asynchronous (Orlander et al., 1980; Orlander et al., 1977; Henriksson and Reitman, 1977). Examination of the time course of training, as well as comparison between training and detraining responses, have led to this conclusion. Several studies have reported a more rapid return of oxidative enzyme activity than $\dot{V}O_2$ max to pre training levels following short periods of detraining, indicating that the oxidative potential of local muscle is not a major determinant of $\dot{V}O_2$ max (Houston et al., 1979). The return of SDH activity to its initial value following 9 weeks of detraining in the present investigation supports this theory.

The dissociation of systemic and local training effects may reflect the ability to perform at different work intensities. Holloszy (1967) suggested that cardio-vascular adaptation is responsible for increases in maximal aerobic capacity, and that the capacity for prolonged submaximal exercise may be determined by the oxidative capacity of local muscle. Anaerobic threshold, proposed as a criterion measure of submaximum fitness (Ivy et al., 1980; Weltman et al., 1978a) has also been related to the oxidative potential of muscle (Ivy et al., 1980; Rusko et al., 1980).

Recent research indicates that the beneficial effects of training on submaximal performance are not solely incidental to elevations in $\dot{V}O_2$ max, but result from changes specific to submaximal exercise. AT has been significantly correlated with several measures of the oxidative capacity of muscle: Ivy et al. (1980) found a correlation of .91 between ATml and muscle respiratory capacity; Rusko et al. (1980) reported values of .63 and .58 between AT-$\dot{V}O_2$ and ATml and SDH and CS activities. The correlation of .56 found between SDH activity and ATml in this study is similar (Table 4.8). Correlations between ATml and $\dot{V}O_2$ max have varied between .52 and .91 in several studies (Davis et al., 1979; Davis et al., 1976; Rusko et al., 1980, Ivy et al., 1980). The value of .62 reported in this study approximates those found in the literature.

Differences in the rate of decline of $\dot{V}O_2$ max and AT after 3 weeks of detraining support Weltman's (1978a) contention that although there is some commonality between the parameters they are measures of different physiological phenomena. $\dot{V}O_2$ max was not significantly different from the post test until 6 weeks of detraining had passed, yet AT-PO had declined significantly by the third week. This may reflect the close association between AT and local oxidative enzymes.

TABLE 4.8

CORRELATION BETWEEN LOCAL MUSCLE AND SYSTEMIC PARAMETERS BEFORE TRAINING (n = 11)

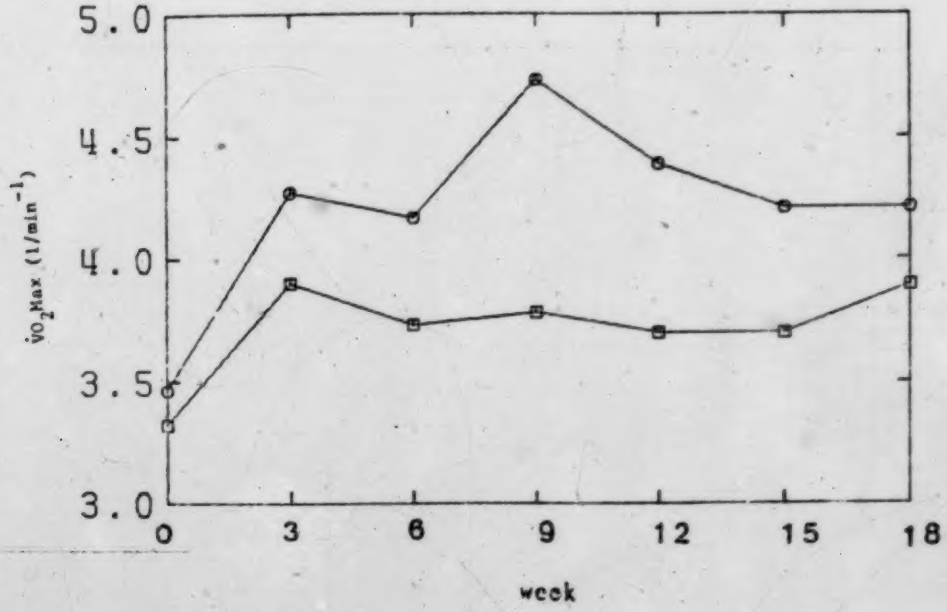
	SDH	% ST	$M\dot{V}O_2$	Atml	HR
SDH	1.00	-0.33	-0.20	0.56	0.12
% ST		1.00	0.12	0.23	0.10
$M\dot{V}O_2$			1.00	0.62	-0.46
ATml				1.00	0.17
HR					1.00

Several mechanisms may be responsible for the large increase in AT, and the subsequent beneficial effects to endurance performance, which occur with training. Intracellular levels of Pi, ADP, and AMP control the rate of glycolysis and glycogenolysis (Holloszy, 1975). Lower steady state concentrations of the phosphates in endurance trained subjects, as the result of decreased lactate accumulation, results in slower rates of glycogenolysis, thus increasing the AT. Evidence of this alteration is seen in the glycogen sparing effect known to occur with training. Increased reliance on lipid oxidation, and the concomitant decrease in glycolysis and lactate accumulation, has also been cited as a mechanism by which AT is increased with training. The drop in RQ often seen following chronic exercise indicates that this is a possible mechanism (Davis et al., 1979). Increased AT-$\dot{V}O_2$ has also been brought about by elevation of blood free fatty acids, and increased shunting of pyruvate to alanine in trained individuals (Ivy et al., 1980). Davis et al. (1979) have suggested that increased muscle blood flow and alteration of fiber recruitment patterns more toward Type I fibers may also be responsible for elevations in AT with training.

Examination of variables representative of the local and central response to chronic exercise and its cessation may increase the understanding of the training stimulus. The knowledge which is derived from time course studies has great application to the specificity of training. The results of the present investigation indicate that the differential physiological changes which occur in response to training and detraining may be representative of functional differences in maximal and endurance performance.

Figure 1. Changes in Mean In Maximal Oxygen Intake (l/min^{-1}) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining

Figure 2. Changes in Mean in Maximal Oxygen Intake (ml.kg.min^{-1}) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining


V̇O2Max (l/min-1)
5.0
4.5
4.0
3.5
3.0
0
3
6
9
12
15
18
week

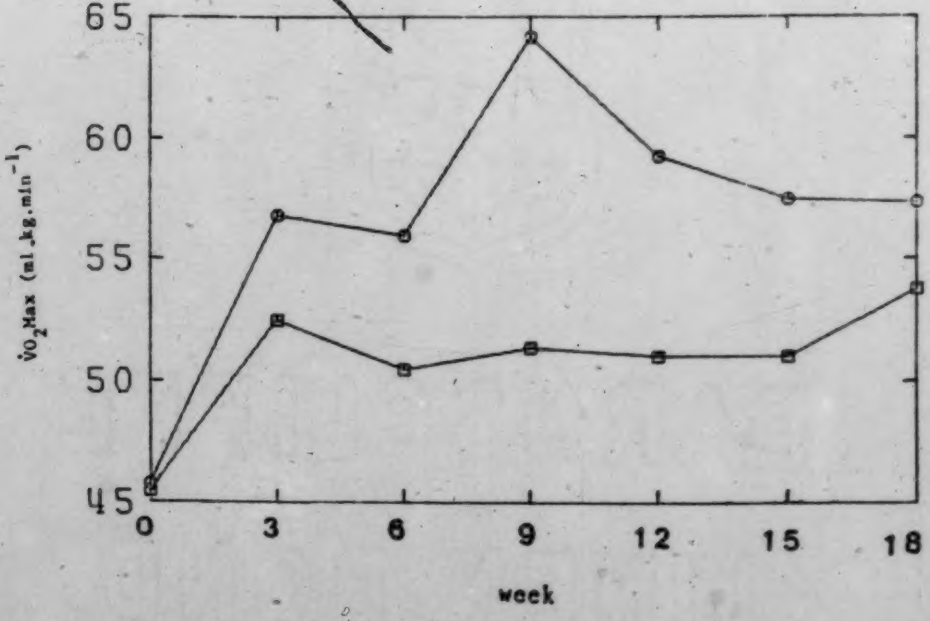
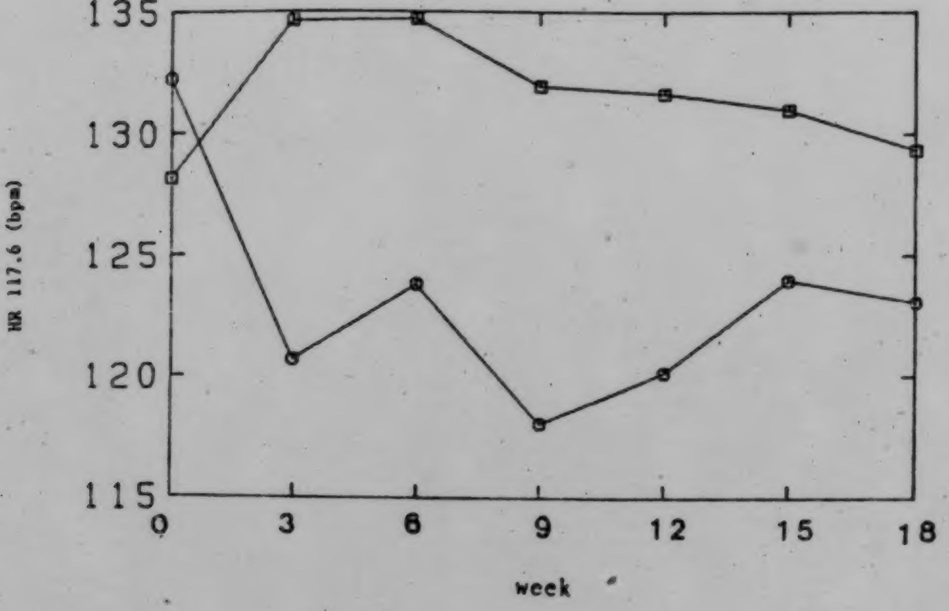

V̇O2Max (ml.kg.min-1)
65
60
55
50
45
0
3
6
9
12
15
18
week

Figure 3. Changes in Mean in Submaximal Heart Rate (bpm) of Control(□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining

Figure 4. Changes in Mean in Anaerobic Threshold (% of $\dot{V}O_2$max) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining


HR 117.6 (bpm)
135
130
125
120
115
0
3
6
9
12
15
18
week

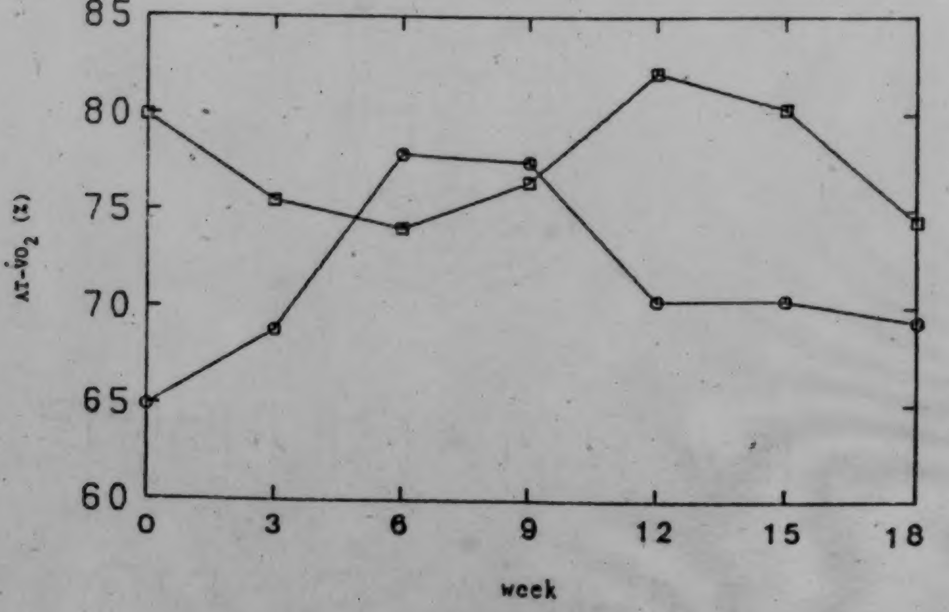
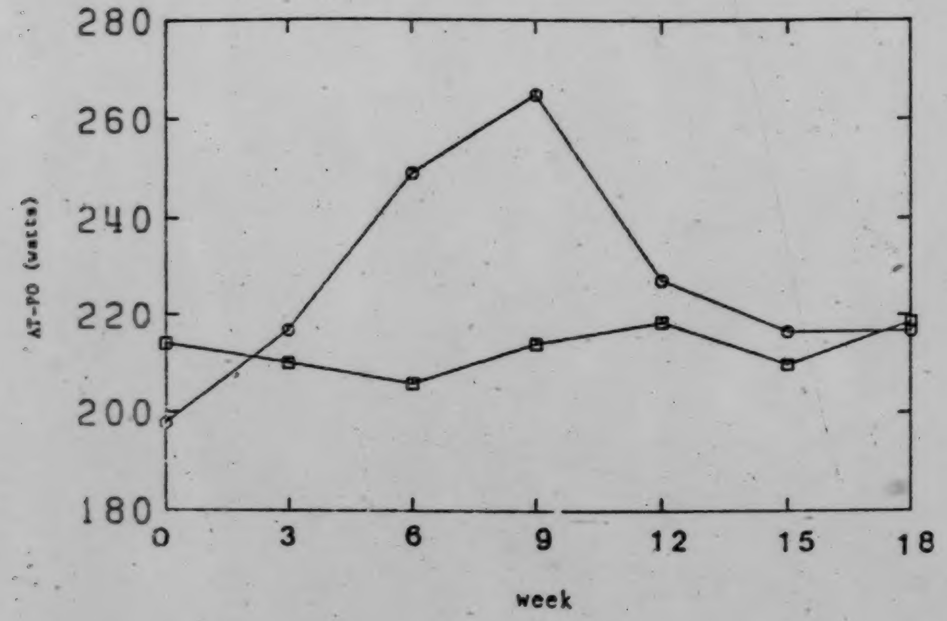

AT-$\dot{V}O_2$ (%)
85
80
75
70
65
60
0
3
6
9
12
15
18
week

Figure 5. Changes in Mean in Anaerobic Threshold (watts) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining

Figure 6. Changes in Mean in Anaerobic Threshold ($\dot{V}O_2$-ml/kg.min^{-1}) of Control (□) and Experimental (O) Males Subjects During 9 Weeks of Training and 9 Weeks of Detraining


AT-PO (watts)
280
260
240
220
200
180
0
3
6
9
12
15
18
week

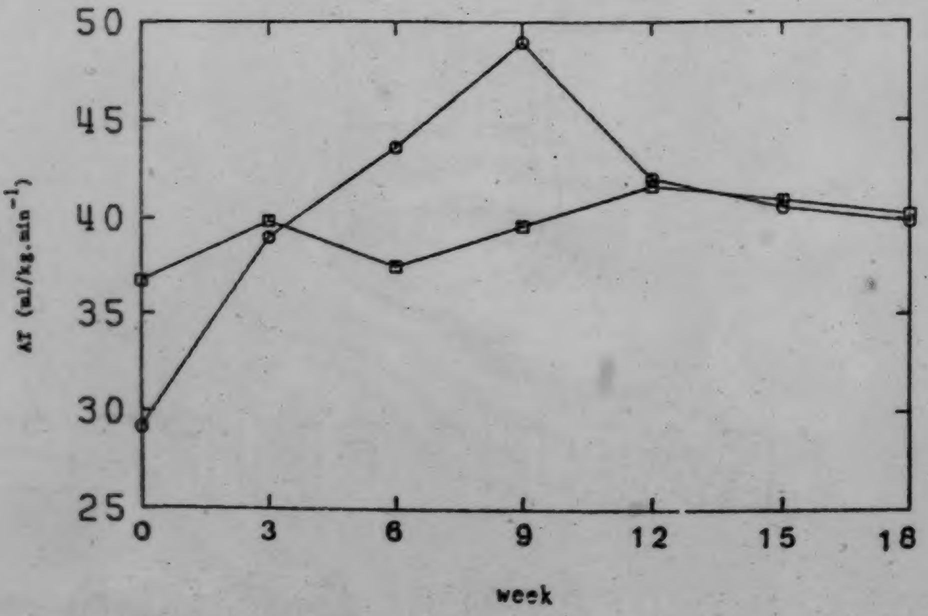
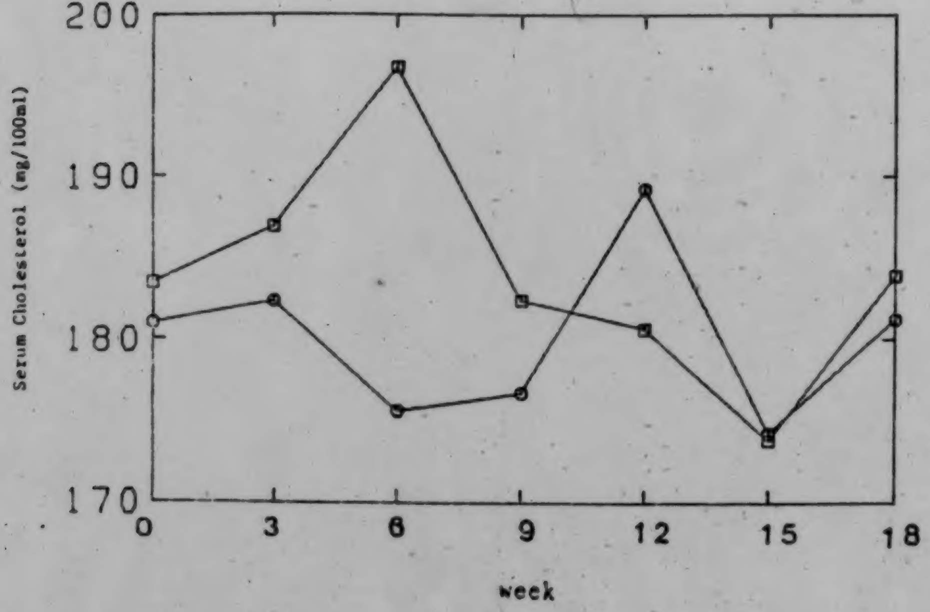

AT (ml/kg.min^{-1})
50
45
40
35
30
25
0
3
6
9
12
15
18
week

Figure 7. Changes in Mean in Total Serum Cholesterol (mg/100ml) of Control (□) and Experimental (O) Males Subjects During 9 Weeks of Training and 9 Weeks of Detraining

Figure 8. Changes in Mean in Serum Triglyceride (mg/100ml) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining


Serum Cholesterol (mg/100ml)
200
190
180
170
0
3
6
9
12
15
18
week

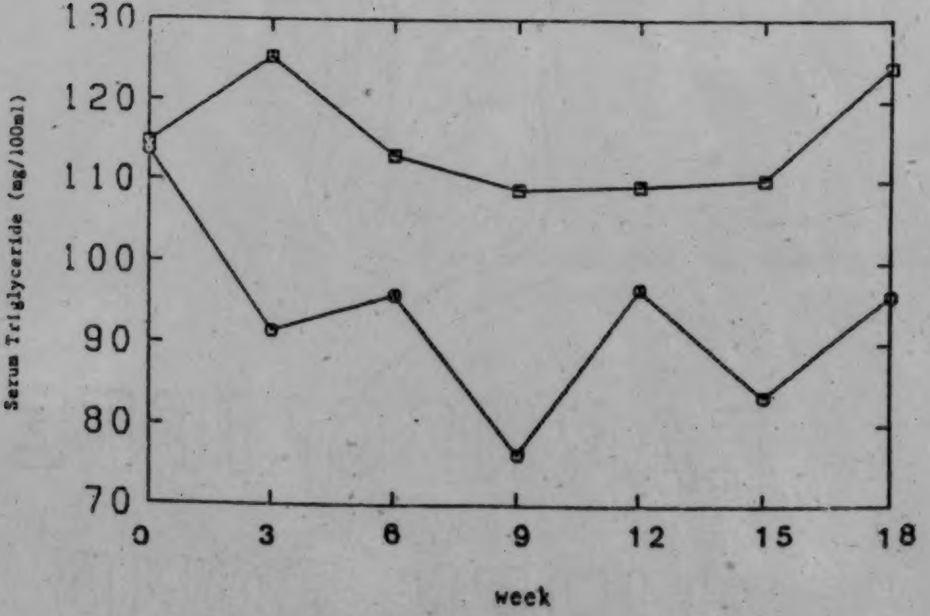
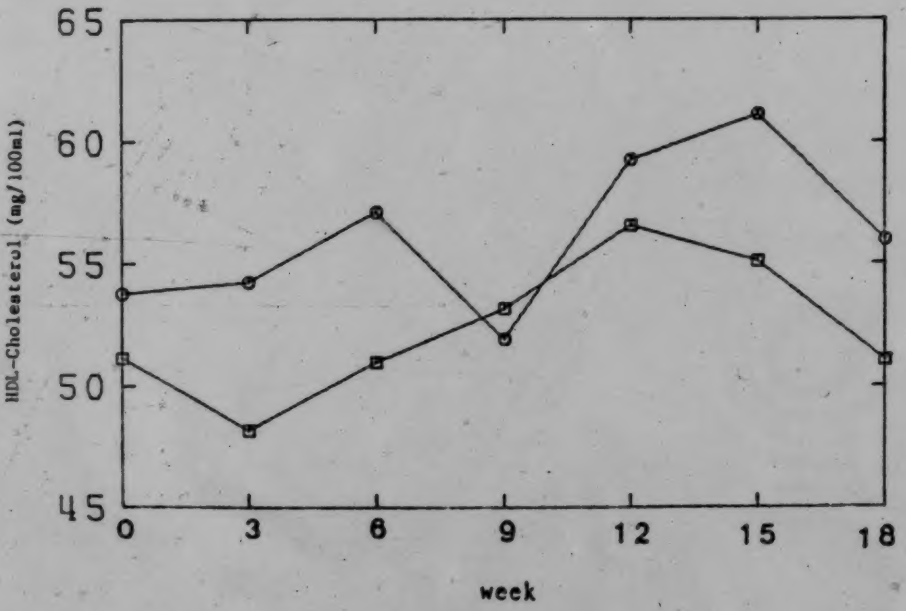
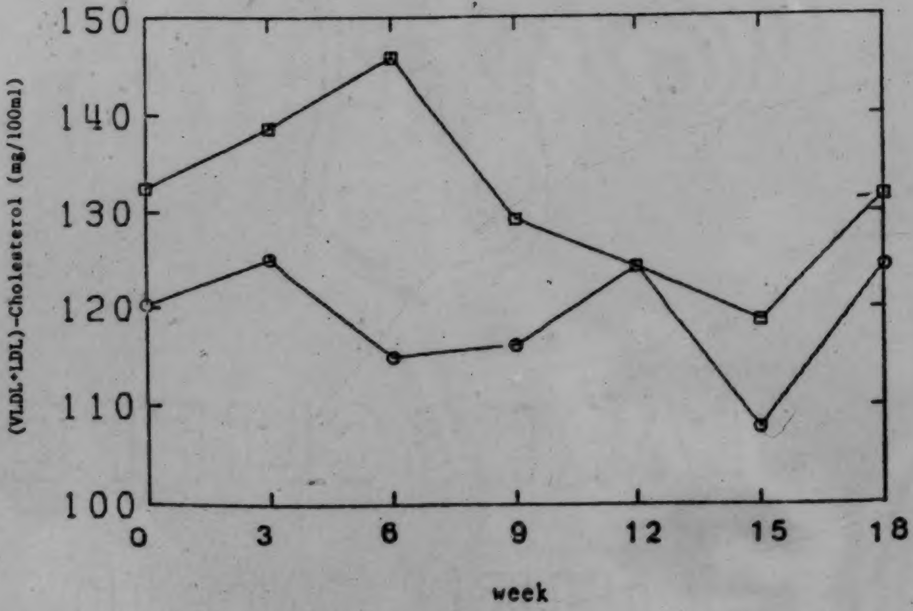

Serum Triglyceride (mg/100ml)
130
120
110
100
90
80
70
0
3
6
9
12
15
18
week

Figure 9. Changes in Mean in Serum HDL-cholesterol (mg/100ml) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining

Figure 10. Changes in Mean in Serum (VLDL + LDL)-cholesterol (mg/100ml) of Control (□) and Experimental (O) Male Subjects During 9 Weeks of Training and 9 Weeks of Detraining


65
60
55
50
45
HDL-Cholesterol (mg/100ml)
0
3
6
9
12
15
18
week


150
140
130
120
110
100
(VLDL+LDL)-Cholesterol (mg/100ml)
0
3
6
9
12
15
18
week

CHAPTER V

SUMMARY AND CONCLUSIONS

The physiological response of 12 male subjects to 9 weeks of endurance training and 9 weeks of detraining was studied. Seven men acted as controls. The experimental and control groups were divided in half and designated as 'hi fit' and 'lo fit'. Bicycle ergometer tests and venous blood samples were performed at 3 week intervals to measure systemic parameters and serum lipids and lipoproteins. Muscle biopsies were taken on three occasions during the investigation.

Significant changes in maximum oxygen intake and anaerobic threshold occurred during the training and detraining periods. Submaximal heart rate decreased significantly with training. Although muscle fiber distribution was not significantly altered as the result of training a trend toward increased FTa and decreased FTb fibers was documented. SDH activity was also elevated non-significantly in the exercise group following training. These changes were reversed during the 9 week detraining period. There were no significant changes in total serum cholesterol, serum triglyceride, serum HDL-cholesterol, serum (VLDL + LDL)-cholesterol, or HDL-cholesterol/total cholesterol during the study.

Within the limitations of the present experiment the following conclusions appear justified:

1. Three weeks of endurance training was sufficient to cause a significant increase in maximal aerobic power and anaerobic threshold.
2. Although substantial losses of training gains in anaerobic

threshold and maximal aerobic power occurred after 3 and 6 weeks of detraining respectively, there was still significant retention after 9 weeks.

3. Changes in maximal aerobic power and anaerobic threshold are partially associated yet do not occur in parallel.
4. Local muscle changes which result from endurance training appear to be reversed within an equal detraining period.
5. Nine weeks of endurance training was not sufficient duration for changes to occur in serum lipids and lipoproteins.
6. Classification of subjects as 'hi fit' and 'lo fit' did not result in a differential response to endurance training and detraining.

Several recommendations for further investigation of the physiological response to training and detraining can be made :

1. Subjects should be placed into distinct high and low fitness groups on the basis of cardiovascular endurance and/or anaerobic threshold.
2. Single subject experimental design may be used to control interindividual variability during examination of the response of serum lipids and lipoproteins to training and detraining.
3. Evaluation of the response of local muscle parameters to training and detraining should be done at more frequent intervals.
4. Longer periods of detraining, and periods of retraining, should be investigated.

5. Comparison of changes with training and detraining in response to programs of interval training and continuous endurance training should be made.
6. The response of anaerobic threshold to training and detraining should be examined in more detail.

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APPENDIX A

CALCULATIONS PERFORMED BY THE METABOLIC MEASUREMENT CART

DATA COLLECTED

The Exercise Metabolic (EM) Program collects the following data from the analyzers in the MMC:

F_{ECO_2}	mixed expired CO_2 (fraction)
F_{EO_2}	mixed expired O_2 (fraction)
Temp	temperature of expired gas as it passes through the volume transducer (°C)
P_B	barometric pressure (mmHg)
Vol	cumulative expired volume (liters, ATPS)
Time	duration of measurement interval (seconds)

Body weight (wt) is entered through the keyboard in pounds or kilograms. When entered in pounds, the calculator converts the entry value to kilograms before storing this value for subsequent calculation.

CALCULATIONS PERFORMED

The Exercise Metabolic Program performs the following calculations using the preceding input data:

Minute Volume (mℓ/min, BTPS)

1. $\dot{V}_{E_{BTPS}} = \text{Vol} \times \frac{60}{\text{Time}} \times \frac{P_B-25}{P_B-47} \times \frac{273^\circ + 37^\circ\text{C}}{\text{Temp} + 273} \times 1000$ (Definition)

2. $= \frac{\text{Vol}}{\text{Time}} \times \frac{P_B-25}{P_B-47} \times \frac{1.86 \times 10^7}{\text{Temp} + 273}$ (Calculation)

3. $\dot{V}_{E_{STPD}} = \dot{V}_{E_{BTPS}} \times \frac{P_B-47}{760 \text{ mmHg}} \times \frac{273^\circ\text{C}}{310^\circ\text{C}}$ (Definition)

4. $= \dot{V}_{E_{BTPS}} \times \frac{P_B-47}{863}$ (Calculation)

Oxygen Consumption (mℓ/min STPD)

5. $F_{IN_2} = 1 - F_{IO_2}$

6. $F_{EN_2} = 1 - F_{EO_2} - F_{ECO_2}$

7. $\dot{V}_{I_{STPD}} = \dot{V}_{E_{STPD}} \times \dfrac{F_{EN_2}}{F_{IN_2}}$

8. $\dot{V}_{O_2} = \left[\dot{V}_{I_{STPD}} \times F_{IO_2}\right] - \left[\dot{V}_{E_{STPD}} \times F_{EO_2}\right]$ (Definition)

Substituting 5 and 6 into 7, and 7 into 8,

9. $\dot{V}_{O_2} = \left[\dot{V}_{E_{STPD}} \times \dfrac{(1-F_{EO_2}-F_{ECO_2})}{1-F_{IO_2}} \times F_{IO_2}\right] - \left[\dot{V}_{E_{STPD}} \times F_{EO_2}\right]$

For: $F_{IO_2} = .2094$, and factoring 9

10. $\dot{V}_{O_2} = \dot{V}_{E_{STPD}} \left\{[.2649 \times (1-F_{EO_2}-F_{ECO_2})] - (F_{EO_2})\right\}$ (Calculation)

If body weight is entered, $\dot{V}_{O_2}$ is also calculated per kg body (mℓ/min/kg)

11. $\dot{V}_{O_2} = \dfrac{\dot{V}_{O_2}}{\text{Wt in kg}}$

Carbon Dioxide Production (mℓ/min, STPD)

12. $\dot{V}_{CO_2} = \left[\dot{V}_{E_{STPD}} \times F_{ECO_2}\right] - \left[\dot{V}_{I_{STPD}} \times F_{ICO_2}\right]$ (Definition)

for low concentrations of inspired CO_2

$\left[\dot{V}_{E_{STPD}} \times F_{IOC_2}\right] - \left[\dot{V}_{I_{STPD}} \times F_{ICO_2}\right]$ is very small and $\dot{V}_{I_{STPD}}$ is a close approximation of $\dot{V}_{E_{STPD}}$

for $F_{ICO_2} = .0003$ (normal air mixtures)

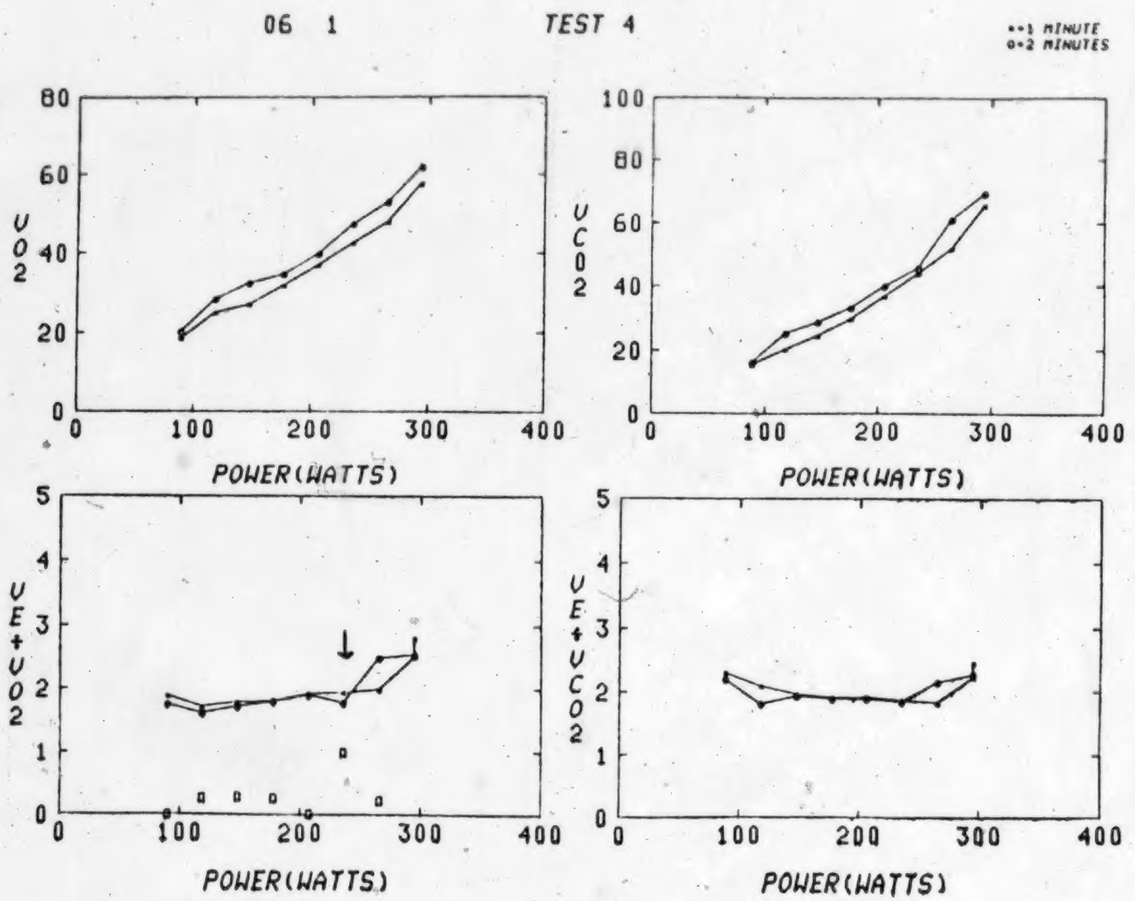
13. $\dot{V}_{CO_2} = \dot{V}_{E_{STPD}} (F_{ECO_2} - .0003)$ (Calculation)

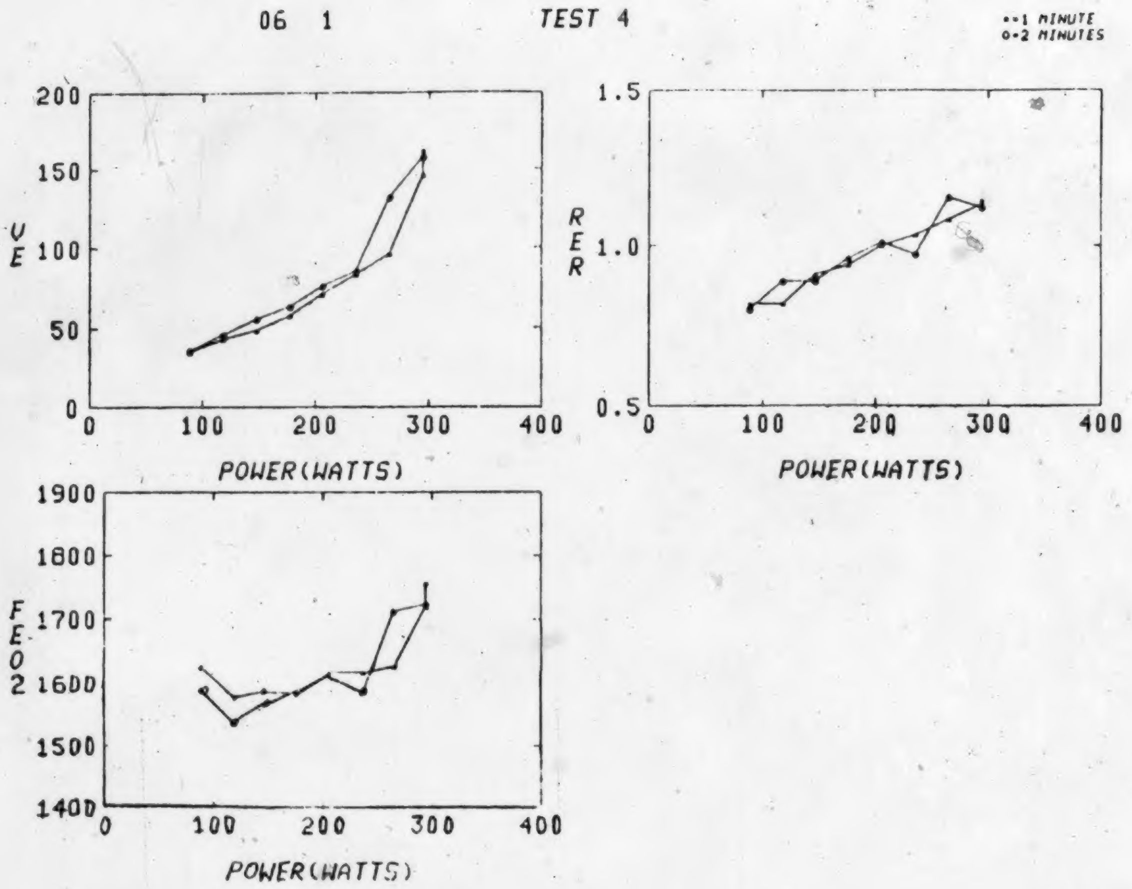
Respiratory Quotient

14. $R = \dfrac{\dot{V}_{CO_2}}{\dot{V}_{O_2}}$

APPENDIX B

GRAPHICAL DETERMINATION OF ANAEROBIC THRESHOLD


06 1
TEST 4
*=1 MINUTE
o=2 MINUTES
VO2
0
20
40
60
80
VCO2
0
20
40
60
80
100
0
100
200
300
400
POWER(WATTS)
VE+VO2
VE+VCO2
0
1
2
3
4
5


06 1
TEST 4
•=1 MINUTE
o=2 MINUTES
VE
200
150
100
50
0
0
100
200
300
400
POWER(WATTS)
RER
1.5
1.0
0.5
0
100
200
300
400
POWER(WATTS)
FEO2
1900
1800
1700
1600
1500
1400
0
100
200
300
400
POWER(WATTS)

APPENDIX C

MYOFIBRILLAR ATPase STAINING PROCEDURE

PREPARATION OF REAGENTS

1. Basic Medium:

Glycine	1.98 g
Calcium Chloride	2.10 g
NaCl	1.45 g
NaOH	0.95 g
Water	500 ml

Mix this solution and store cold. When ready to use adjust the pH using NaOH or HCL (5N) for rinse, preincubations, and incubation media.

2. Acid Preincubation Media:

Sodium Acetate .3H_2O	6.47 g
Kcl	3.70 g
Water	500 ml

The pH of this will be approximately 7. Divide into 3 portions and adjust pH to approximately 4.4, 4.6, and 4.8 with glacial acetic acid.

3. Incubation Medium: (fresh)

Take 10 ml of pH 10.3 preincubation medium
Add 0.017 g ATP (Boehringer 15028)
Mix
Adjust to pH 9.4 with 1N HCL
(Good for 1 day only)

4. $CaCl_2$ Solution (1%):

1 g $CaCl_2$ (72161 Fisher) to 100 ml distilled water

5. $CaCl_2$ Solution (2%):

3.66 g $CaCl_2$ (Merck 2539) to 100 ml distilled water

6. $(NH_4)_2S$ Solution (1%):

20% $(NH_4)_2S$ (Merck 5442) diluted 20 times with distilled water

PROCEDURE

1. Preincubate:	pH 10.30	8 minutes at 37°C water bath
	pH 4.61	50 seconds at room temperature
	pH 4.30	5 minutes at room temperature

2. Rince in pH 9.4 preincubation medium (without ATP) 2 times for 30 seconds.
3. Incubate 30 minutes at pH 9.4 (with ATP solution) at 37°C.
4. Rinse in $CaCl_2$ (1%) solution as follows: 1 minute then empty $CaCl_2$, rinse 2 minutes in new $CaCl_2$, then rinse 3 minutes in new $CaCl_2$. All at room temperature.
5. Rinse in $CaCl_2$ (2%) the same way, 3 times for 1 minute each.
6. Rinse carefully 25 times in distilled water.
7. Put in 1% $(NH_4)_2S$ for 1 minute at room temperature.
8. Rinse 25 times in distilled water.
9. Mount in Permount.

APPENDIX D

NADH-DIAPHORASE STAINING PROCEDURE

PREPARATION OF REAGENTS

1. Tris Buffer (pH 7.4):

Tris Hydroxymethylaminomethane	0.606 g
Distilled Water	58.0 ml
.1 M Hydrochloric Acid	42.0 ml

The pH will be approximately 7.4.

To be made fresh.

2. Nitro Blue Tetrazolium Salt (NBT) M.W. 816.0
3. Reduced diphosphopyridine nucleotide (NADH) M.W. 709.4

PROCEDURE

1. Incubate tissue for 30 minutes at 37°C in the following solution:

0.2 M Tris Buffer (pH 7.4)	10 ml
NBT	10 mg
NADH	8 mg

*The pH is adjusted to 7.4 with HCl or NaOH.

2. Rinse in cool distilled water, 3 x 1 minute.
3. Mount in Permount.

APPENDIX E

HOMOGENIZATION PROCEDURE

Buffer = 0.1 Tris at (6.05g/500 ml) pH 7.5 - stored in fridge.

1. Remove blood and connective tissue from sample while thawing in ice cold Tris buffer.
2. Blot sample and weigh on Mettler to nearest tenth of a milligram.
3. Place sample in glass homogenizer with 0.5 ml buffer. Place homogenizer in an ice water bath. Grind three times for 3-4 seconds in 30 sec intervals. Add another 0.5 ml of buffer and grind twice more. Pour off into test tube. Add another 2 ml buffer to homogenizer, swish around also cleaning pestle and pour into test tube thus diluting sample in 3 ml of buffer.
4. Do enzyme determination.

APPENDIX F

SUCCINATE DEHYDROGENASE BIOCHEMICAL PROCEDURE

		Initial Concentration	Final Concentration
1.	.02 ml of muscle homogenate.		
2.	0.75 potassium phosphate buffer (6.846 g) with .05% BSA (50 mg) in 100 ml H_2O at pH 7.7.	.3M	.2M
3.	Let stand 5 min. at room temperature.		
4.	Add 10 ul phenazine methosulphate - PMS 14 mg/ml	45.6mM	.42mM
5.	Add 140 ul Succinic Acid Disodium Salt (1.6 g/10 ml)	1M	.13M
6.	Incubate exactly 30 min. in dark water bath at 38°C.		
7.	Stop the reaction with 225 ul of 1M NaOH.		
8.	Add 500 ul of stock bromobenzene and mix.		
9.	1825 ul Total Volume.		
10.	Centrifuge at 2000 g for 5 min.		
11.	Add 500 ul supernatant to 2.5 ml of fresh hydrazine buffer in 100 ml (1.3 g) with 2mM EDTA > H_2O 74.5 mg) and 0.4 mM NAD > pH 9.0 (27.6 mg)	.1M 2mM 0.4mM	.083M 1.67mM 0.33mM
12.	Read blank fluorescence.		
13.	Add 5 ul Fumerase - 0.25 ug/ml.		
14.	Add 75 ul malic dehydrogenase - 5 ug/ml.		

Allow reaction to run to completion (approximately 2 hours) and read fluorescence again.

$$\text{Succinate} + \text{FAD} \xleftrightarrow{\text{SDH}} \text{Fumerate} + \text{FADH}_2$$

$$\text{Fumerate} + H_2O \xleftrightarrow{\text{fumerase}} \text{Malate}$$

$$\text{Malate} + \text{NAD} \xleftrightarrow{\text{MDH}} \text{Oxaloacetate} + \text{NADH} + H^+$$

SAMPLE SDH CALCULATION

WET WEIGHT of muscle = ____________ mg

Volume buffer = ____________ ml

1 UNIT Δ F = .00008 μmoles NADH/ml

NET change = ____________ UNITS

therefore

Tissue caused change = ____________ x .00008 = __________ moles NADH/ml

Homogenate dilution = ____________ mg/____________ ml

= ____________ mg/ml

TOTAL volume of 1st Rx. mix = 6.58 ml

Quantity of muscle in 1st Rx. mix = .08 x __________ (H. D.)

= __________ mg

[muscle sample] in 1st Rx. mix = __________ mg/6.58 ml

= __________ mg/ml

Final Rx. volume = 3080μl

Quantity of muscle = 500μl of 1st Rx. mix

= .5 x _________ [1 Rx. muscle]

= ___________ mg tissue

muscle sample in final Rx. = ___________ mg/3.08 ml

= ___________ mg/ml

____________ mg tissue caused a change equivalent to

____________ μmoles NADH/ml in 30 minutes

__________ μmoles/ ___________ mg/30 min

__________ μmoles/ ___________ mg/min

__________ μmoles/ g / minute

APPENDIX G

CALCULATION OF PER CENT BODY FAT

MEASUREMENTS:

(1) Wt. in air ________ (lbs.)

(2) Vital capacity (V.C.) ________ (liters) x 61.02 = ________ (cu.in.)

(3) Residual Volume 25% of V.C. = ________ (cu.in.)

(4) Vol. Gastro-intestinal track (VGI) = 7.01 (cu.in.)

(5) Wt. in water (full inspiration)

Wt. in water $= \left[\frac{\text{Chart Reading x } 18.08}{75}\right] - 18.08$ (lbs.) = ________ (must be negative)

CALCULATIONS:

(6) Total body air (T.B.A.) = V.C. ________ (cu.in.) (from 2 above)

+ R.V. ________ (cu.in.) (from 3 above)

+ RGI 7.01 (cu.in.)

= ________ x 0.362 = ________ (lbs.)

(7) True wt. in water = weight in water (from 5 above) ________ (lbs.)

+ total body air (from 6 above) ________ (lbs.)

= ________ (lbs.)

(8) Body Volume = wt. in air (1) ________ - true wt. in water

(7) ________ = ________ (lbs.)

(9) Body density $= \left[\frac{\text{wt. in air (1) \_\_\_\_\_\_}}{\text{Body volume (8) \_\_\_\_\_\_}}\right]$ X density of H_2O ________

= ________

(10) % Fat $= \left[\frac{4.570}{\text{Body Density}} - 4.142\right] \times 100$

= ________ %

(11) Lbs. fat = [________ (% fat) x ________ (wt. in air)] ÷ 100

= ________ (lbs.)

(12) Lbs. fat free wt. = ________ wt. in air (1) - lbs. fat (11) ________

= ________ (lbs. fat free wt.)

APPENDIX H

SAMPLE DIET ANALYSIS

```
15/ 7/80  HELLO                          . HERE IS YOUR PERSONALIZED
NUTRITION EVALUATION BASED ON THE INFORMATION YOU REPORTED.

CANADIANS ARE GENERALLY EATING POORLY, ACCORDING TO STUDIES DONE BY
NUTRITION CANADA AND OTHERS. HERE IS AN OPPORTUNITY FOR YOU TO EVALUATE
YOUR OWN DIET AND THEREBY GET A BETTER UNDERSTANDING OF THE AMOUNT AND
VARIETY OF FOODS YOU NEED TO MAINTAIN GOOD HEALTH.

                          YOUR PERSONAL DATA
-----------------------------------------------------------------------
THE PERSONAL DATA YOU REPORTED IS SHOWN BELOW. THE COMPUTER HAS IDENTI-
FIED THE IDEAL WEIGHT RANGE FOR A PERSON OF YOUR AGE AND SEX.
-----------------------------------------------------------------------
-  FEMALE,  BIRTH DATE 18/ 2/53, AGE  27, HEIGHT 167 CM ( 66 INS.),     -
-            WEIGHT  57 KG ( 128 LBS.), MEDIUM FRAME SIZE               -
- THE AVERAGE WEIGHT RANGE FOR YOU IS  57 TO  64 KG ( 128 TO 143 LBS.) -
-----------------------------------------------------------------------

                          YOUR ACTIVITY DATA
-----------------------------------------------------------------------
THE ACTIVITY DATA YOU REPORTED HAS BEEN AVERAGED. THE CALORIE EQUIVALENT
FOR AN AVERAGE PERSON IS SHOWN FOR THE DIFFERENT ACTIVITY LEVELS.
-----------------------------------------------------------------------
- LEVEL 1    6.67 HOURS RESTING            REQUIRES        358 CALORIES -
- LEVEL 2   14.83 HOURS SEDENTARY          REQUIRES       1068 CALORIES -
- LEVEL 3    1.33 HOURS LIGHT ACTIVITY     REQUIRES        200 CALORIES
- LEVEL 4    1.17 HOURS ACTIVE             REQUIRES        315 CALORIES -
-                        TOTAL CALORIC EXPENDITURE =  1941 CALORIES -
-----------------------------------------------------------------------

CALORIES ARE A UNIT OF MEASURE FOR ENERGY IN THE SAME WAY THAT INCHES
ARE A MEASURE FOR LENGTH. YOUR SIZE, BOTH HEIGHT AND WEIGHT, AND YOUR
ACTIVITY LEVEL DETERMINE HOW MANY CALORIES YOU USE EACH DAY. IF YOU
PROVIDE YOUR BODY WITH THE SAME NUMBER OF CALORIES THAT YOU USE UP YOUR
WEIGHT WILL REMAIN CONSTANT. WEIGHT GAIN OCCURS WHEN THE BODY HAS TO
STORE EXCESS CALORIES (UNUSED ENERGY) AND WEIGHT LOSS OCCURS WHEN THE
BODY HAS TO USE STORED ENERGY.

                          YOUR CALORIE BREAKDOWN
-----------------------------------------------------------------------
YOUR CALORIES COME FROM THE FOLLOWING SOURCES, AS SHOWN IN THE TABLE
BELOW, AND ARE EXPRESSED AS AN APPROXIMATE PERCENTAGE OF YOUR TOTAL
CALORIC INTAKE. THE IDEAL BALANCE FOR CALORIE SOURCES IS GIVEN.
-----------------------------------------------------------------------
              CARBOHYDRATE                  FAT +                PROTEIN
-----------------------------------------------------------------------
- YOURS           45.7                      40.1                 12.5  -
- IDEAL           53.0                      35.0                 12.0  -
-----------------------------------------------------------------------
```

```
THAN 35% OF YOUR TOTAL CALORIES TO HELP PREVENT HEART DISEASE. REFER TO
'GOOD EATING TO GUARD YOUR HEART' AVAILABLE FROM YOUR LOCAL HEALTH UNIT.

                         YOUR FOOD GROUPS
-------------------------------------------------------------------------
THE FOOD AND DRINK ITEMS YOU REPORTED HAVE BEEN CATEGORIZED INTO THE SIX
BASIC FOOD GROUPS. THE FOLLOWING TABLE COMPARES THE NUMBER OF SERVINGS
YOU NEED DAILY WITH THE AVERAGE NUMBER OF SERVINGS IN YOUR DIET.
-------------------------------------------------------------------------
          FOOD                     NO. OF RECOMM.      AVERAGE NUMBER OF
         GROUPS                       SERVINGS          SERVINGS YOU HAD
-------------------------------------------------------------------------
- 1. GRAINS, BREADS & CEREALS        3.0 TO 5.0             2.7      -
- 2. MILK AND MILK PRODUCTS          2.0 TO 2.0             2.4      -
- 3. MEAT AND ALTERNATIVES           2.0 TO 2.0             1.4      -
- 4. FRUIT AND VEGETABLES            4.0 TO 5.0             5.7      -
- 5. FAT AND OILS                                           9.3      -
- 6. SWEETS AND DESSERTS                                    9.3      -
-------------------------------------------------------------------------

                      YOUR NUTRIENT BREAKDOWN
-------------------------------------------------------------------------
THE FOOD AND DRINK ITEMS YOU REPORTED HAVE BEEN SEPARATED INTO THE FOOD
COMPONENTS SHOWN BELOW. THE RECOMMENDED AMOUNTS FOR WEIGHT MAINTENANCE
FOR A PERSON OF YOUR SEX, AGE AND ACTIVITY ARE COMPARED TO YOUR INTAKE.
-------------------------------------------------------------------------
  FOOD OR               RECOMMENDED  YOUR INTAKE    % OF    INTAKE LESS
  NUTRIENT    UNIT        AMOUNT       AMOUNT      RECCM.   THAN RECOM.
-------------------------------------------------------------------------
- CALORIES    KCAL        1938.0       2313.5      119                 -
- PROTEIN       GM          41.6         72.5      174                 -
- THIAMIN       MG           1.1          1.5      133                 -
- NIACIN        MG          14.0         29.9      213                 -
- RIBOFLAVIN    MG           1.3          1.9      143                 -
- VIT. B6       MG           1.5          1.5      102                 -
- FOLATE       MCG         200.0        215.4      107                 -
- VIT. B12     MCG           3.0          2.4       80        YES      -
- VIT. C        MG          30.0        134.3      447                 -
- VIT. A        RE         800.0       1129.6      141                 -
- CALCIUM       MG         700.0       1206.1      172                 -
- PHOSPHORUS    MG         700.0       1551.8      222                 -
- IRON          MG          14.0         14.8      105                 -
- PANTOTHENICA**MG           5.0          8.4                          -
- SODIUM      **GM        2 TO 8          2.3                          -
- FIBRE       **GM        5 TO 8          6.3  ** SUGGESTED VALUES AS NO -
- CARBOHYDRATE GM                       264.1     STANDARDS HAVE BEEN SET-
- FAT           GM                      103.2                          -
- ALCOHOL       GM                       16.0                          -
-------------------------------------------------------------------------

IT IS USUAL FOR YOUR NUTRIENT INTAKE TO VARY FROM DAY TO DAY. SOME ARE
STORED AND ONLY REQUIRE AN ADEQUATE WEEKLY INTAKE, SUCH AS IRON AND VITA-
MIN A. OTHERS, LIKE VITAMIN C, ARE NOT STORED AND ARE NEEDED DAILY. IT IS
BEST TO HAVE AN ADEQUATE SUPPLY OF ALL NUTRIENTS  ON A DAILY BASIS.
```

```
                      YOUR DAILY NUTRIENT BREAKDOWN
------------------------------------------------------------------------
FOOD OR                      DAY  #1#        DAY  #2#         DAY  #3#
NUTRIENT   UNIT            AMOUNT  %       AMOUNT  %        AMOUNT  %
------------------------------------------------------------------------
CALORIES   KCAL            2160.5 111       2376.4 122       2403.6 124
PROTEIN      GM              66.1 158         67.4 161         83.9 201
THIAMIN      MG               1.1  96          1.6 144          1.7 156
NIACIN       MG              23.9 170         33.8 241         32.1 228
RIBOFLAVIN   MG               1.6 122          1.5 116          1.9 143
VIT. B6      MG               1.1  71          1.7 113          1.8 112
FOLATE      MCG             186.1  93        252.2 126        207.9 103
VIT. B12    MCG               2.4  75          1.7  53          3.2 [illegible]
VIT. C       MG              35.1 283        136.6 456        120.9 403
VIT. A       RE             773.7  96       1671.3 208        943.8 117
CALCIUM      MG            1175.5 167        803.3 114       1639.4 234
PHOSPHORUS   MG            1297.1 185       1371.2 195       1996.2 285
IRON         MG              13.6  97         14.9 106         15.9 113
PANTOTHENIC**MG               8.8             10.4              6.1
SODIUM     **GM               3.5              1.4              2.7
FIBRE      **GM               5.3              7.4              7.0
CARBOHYDRATE GM             233.7            265.4            293.3
FAT          GM              92.3            106.8            110.5
ALCOHOL      GM              27.0             27.0              0.0
------------------------------------------------------------------------

          YOUR DAILY INTAKE BY FOOD GROUPS IN SERVINGS PER DAY
------------------------------------------------------------------------
 FOOD GROUPS               DAY  #1#          DAY  #2#          DAY  #3#
------------------------------------------------------------------------
GRAINS,BREAD,ETC            3.00             1.00              4.00
MILK & MILK PROD            2.60             1.00              3.65
MEAT & ALTERNATE            0.75             2.15              1.23
FRUIT & VEGETABL            4.00             6.10              7.00
FATS AND OILS               8.00             8.50             11.50
SWEETS & DESSERT            9.00            13.75              5.25
------------------------------------------------------------------------

                          YOUR DAILY ACTIVITY
------------------------------------------------------------------------
 ACTIVITY                  DAY  #1#          DAY  #2#          DAY  #3#
------------------------------------------------------------------------
INACTIVE                    6.00             8.00              6.00
NOT VERY ACTIVE            16.00            13.50             15.00
SLIGHTLY ACTIVE             1.00             1.00              2.00
ACTIVE                      1.00             1.50              1.00
                         RECOMMENDATIONS
                         ---------------

** YOUR CALORIC INTAKE IS HIGH FOR YOUR STATED LEVEL OF ACTIVITY AND MAY
   CAUSE YOU TO GAIN WEIGHT IF CONTINUED ON A REGULAR BASIS.

** WE SUGGEST YOU CHOOSE MORE FOODS CONTAINING VITAMIN B12
```

** PROTEINS ARE MADE UP OF A NUMBER OF AMINO ACIDS. THE AMINO ACIDS THAT CANNOT BE SYNTHESIZED IN THE BODY MUST ALL BE PRESENT IN YOUR DIET ON A DAILY BASIS BECAUSE THEY ARE REQUIRED FOR THE GROWTH OF NEW TISSUE, REPAIR OF OLD TISSUE AND REGULATION OF IMPORTANT BODY FUNCTIONS. THE DEMAND FOR ENERGY TAKES FIRST PRIORITY IN METABOLISM. IF CARBOHYDRATE AND FAT ARE NOT CONSUMED IN SUFFICIENT AMOUNTS SOME OF THE AMINO ACIDS WILL BE USED AS A SOURCE OF ENERGY AND WILL NOT BE AVAILABLE FOR THE SYNTHESIS OF BODY PROTEINS. WHEN ENERGY CONSUMPTION IS LOW PROTEIN IS USED LESS EFFICIENTLY. THE CORRECT BALANCE BETWEEN PROTEIN, FAT AND CARBOHYDRATE INTAKE IS VERY IMPORTANT. THE RECOMMENDATION FOR PROTEIN INTAKE IS CLOSE TO DOUBLE THE AVERAGE REQUIREMENT FOR SOMEONE YOUR AGE AND WEIGHT TO INSURE THAT VARIATIONS IN INDIVIDUAL NEEDS ARE MET.

** CARBOHYDRATES SUPPLY THE MOST EFFICIENT SOURCE OF ENERGY FOR YOUR BODY. MOST OF THE CARBOHYDRATES IN YOUR DIET COME FROM STARCHES AND SUGARS. IT IS HEALTHIER TO CONSUME MOST OF YOUR CARBOHYDRATES AS STARCHES BECAUSE THESE FOODS ALSO CONTAIN MANY NECESSARY VITAMINS AND MINERALS AS WELL AS FIBER. CEREALS OR WHOLE GRAINS, LEGUMES, FRUITS AND VEGETABLES ARE NOURISHING SOURCES OF CARBOHYDRATES. THAT PORTION OF YOUR DIET WHICH COMES FROM SUGARS IS NOTED IN FOOD GROUP 6. A SERVING SIZE IS EQUAL TO 1 TEASPOON OF SUGAR AND IS APPROXIMATELY 15 NON-NOURISHING CALORIES. THE FOODS YOU HAVE EATEN THAT ARE PARTICULARLY HIGH IN SUGAR ARE NOTED IN THE FOOD INPUT LIST.

** FATS SUPPLY THE MOST CONCENTRATED SOURCE OF ENERGY FOR YOUR BODY AND ARE REQUIRED AS A SOURCE OF ESSENTIAL FATTY ACIDS, PARTICULARLY LINOLEIC ACID, AND AS A CARRIER OF THE FAT SOLUBLE VITAMINS A, D, E AND K. THE TYPE AND AMOUNT OF FAT YOU EAT IS IMPORTANT TO YOUR HEALTH AND MANY HEALTH PROFESSIONALS ENCOURAGE A MINIMUM INTAKE OF FAT FROM ANIMAL FOOD SOURCES BALANCED BY SOME FAT FROM VEGETABLE FOOD SOURCES. THAT PORTION OF YOUR DIET WHICH COMES FROM FATS IS NOTED IN FOOD GROUP 5. EACH UNIT IS EQUAL TO THE FAT CONTAINED IN 1 TEASPOON OF BUTTER AND CONTAINS APPROXIMATELY 45 CALORIES. FOR MORE INFORMATION PLEASE REFER TO 'GOOD EATING TO GUARD YOUR HEART'.

** VITAMINS AND MINERALS, GENERALLY SPEAKING, ARE SPECIAL SUBSTANCES THAT ARE NEEDED IN SMALL AMOUNTS BY YOUR BODY TO PERFORM COMPLEX CHEMICAL REACTIONS THAT ARE VITAL TO ITS PROPER FUNCTIONING AND HEALTH. VITAMINS PLAY THEIR MOST IMPORTANT ROLE BY INSURING THAT OTHER NUTRIENTS ARE USED EFFECTIVELY. MINERALS ACT AS BODY REGULATORS AND AS BUILDING MATERIALS FOR BOTH HARD AND SOFT TISSUES. THE PAMPHLET 'FUNCTIONS AND SOURCES OF NUTRIENTS IN FOODS', THAT COMES WITH THIS PRINTOUT, IS MORE SPECIFIC. NUTRIENTS ARE INTERRELATED AND THE PROPER BALANCE MUST BE MAINTAINED. EXCESS, AS WELL AS DEFICIENCY, OF ANY NUTRIENT MAY BE HARMFUL. IN OTHER WORDS, A CERTAIN AMOUNT OF EACH NUTRIENT IS ESSENTIAL FOR GROWTH AND MAINTENANCE OF HEALTH; TOO LITTLE CAN CAUSE DEFICIENCY DISEASES AND TOO MUCH CAN PRODUCE TOXICITY OR METABOLIC DISTURBANCES. IT IS IMPORTANT TO KEEP THIS IN MIND BEFORE SELF PRESCRIBING ANY VITAMIN, MINERAL OR OTHER NUTRIENT SUPPLEMENT. A DAILY DIET THAT CONTAINS A VARIETY OF DIFFERENT FOODS FROM WITHIN EACH OF THE IMPORTANT FOOD [illegible] YOU THE [illegible] BALANCE OF ALL NUTRIENTS

```
                               FOOD INPUT LIST
                               ---------------

                                    DAY 1
-------------------------------------------------------------------------------
AMOUNT SERVING SIZE            FOOD NAME                      COMMENT
-------------------------------------------------------------------------------
 2.00  1 OZ          CHEESE,CHEDDAR,HARD
 1.00  1 CUP, 8 OZ   MILK-2%                      VITAMIN D SOURCE
 2.00  1 SLICE       BREAD,100% WHOLE GRAIN
 1.00  1 SLICE       BREAD,WHITE ENRICHED
 0.50  1/4 CUP       RAISINS                      HIGH CALORIC CONTENT
 2.00  1 CUP         SALAD,MIXED GREEN
 1.00  1/2 CUP       POTATO SALAD,DRESSING
 0.50  1 MEDIUM      TOMATOE,RAW
 1.00  2 OZ,2 TH.SL  BEEF/VEAL,ROAST              FAT CONTENT VARIES
 0.50  2 OZ          HAM/BACKBACON                HIGH FAT CONTENT
 1.00  1 TSP, 1 PAT  BUTTER                       HIGH FAT CONTENT
 2.00  1 TBSP,1 PKG  COFFEE CREAMER               HIGH CALORIC CONTENT
 3.00  3 OZ          WINE,TABLE
 3.00  1 CUP         COFFEE
 3.00  1 TSP         SUGAR,WHITE/BROWN            HIGH SUGAR CONTENT
 2.00  1             CAKE,BROWNIES,SQUARES        HIGH SUGAR AND FAT
 2.00  2             COOKIE,SUGAR,ASSORTED        HIGH CALORIC CONTENT
 2.00  1/2 CUP       SOUP,CLAM CHOWDER            RECIPE VARIES
-------------------------------------------------------------------------------

                                    DAY 2
-------------------------------------------------------------------------------
AMOUNT SERVING SIZE            FOOD NAME                      COMMENT
-------------------------------------------------------------------------------
 1.00  1 CUP, 8 OZ   MILK-2%                      VITAMIN D SOURCE
 1.00  1/2 CUP       RICE,BROWN,CKD.
 0.25  1/2 CUP       APPLESAUCE,SW.               HIGH SUGAR CONTENT
 0.50  2             DATES/FIGS                   HIGH SUGAR CONTENT
 1.00  1/4 CUP       RAISINS                      HIGH CALORIC CONTENT
 1.00  1/2 CUP       ORANGE JUICE,UNSW.
 0.50  6 SPEARS      ASPARAGUS,CKD.
 1.00  1 MEDIUM      CARROTS,CKD.
 0.25  1/4 CUP       BEANSPROUTS,FRESH
 2.00  1 CUP         SALAD,MIXED GREEN
 2.00  2 OZ,1 TH SL  PORK ROAST
 2.00  1/4 CUP       PEANUTS,ROASTED              FAT & INCOMPLETE PROTEIN
 0.50  2 TBSP        SEEDS,SUNFLOWER/SESAME       FAT & INCOMPLETE PROTEIN
 4.00  1 TBSP,1 PKG  COFFEE CREAMER               HIGH CALORIC CONTENT
 1.00  2 TBSP        WHITE SAUCE/GRAVY            HIGH CALORIC CONTENT
 1.00  1 TBSP        DRESSING,FRENCH/OIL&VIN      HIGH FAT CONTENT
 3.00  3 OZ          WINE,TABLE
 4.00  1 CUP         COFFEE
 3.00  1 TSP         SUGAR,WHITE/BROWN            HIGH SUGAR CONTENT
 2.00  2 IN. SQUARE  CAKE WITH ICING              HIGH SUGAR AND FAT
 1.00  2             COOKIE,SUGAR,ASSORTED        HIGH CALORIC CONTENT
-------------------------------------------------------------------------------
```

```
                                  DAY 3
-------------------------------------------------------------------------------
AMOUNT SERVING SIZE           FOOD NAME                       COMMENT
-------------------------------------------------------------------------------
 0.25  1 OZ             CHEESE,CHEDDAR,HARD
 0.50  1 OZ             CHEESE,SWISS/GOUDA
 3.00  1 CUP, 8 OZ      MILK-2%                     VITAMIN D SOURCE
 3.00  1 SLICE          BREAD,100% WHOLE GRAIN
 1.00  1                MUFFIN,WHOLE GRAIN,BRAN
 0.25  2                DATES/FIGS                  HIGH SUGAR CONTENT
 3.00  1/4 CUP          RAISINS                     HIGH CALORIC CONTENT
 2.00  1/2 CUP          ORANGE JUICE,UNSW.
 0.50  1/2 CUP          BEANS,GREEN/YELLOW,CKD.
 0.50  1 STALK          BROCCOLI,CKD.
 0.25  1 MEDIUM         CARROTS,CKD.
 0.25  1 CUP            CAULIFLOWER,CKD.
 0.25  1/2 CUP          ONIONS,CKD.
 0.25  1/2 CUP          PEAS,CKD.
 0.25  2 OZ             HAM/BACKBACON               HIGH FAT CONTENT
 4.00  1 OZ             MEATS,DELI TYPE             HIGH FAT CONTENT
 1.00  1/4 CUP          PEANUTS,ROASTED             FAT & INCOMPLETE PROTEIN
 1.00  2 TBSP           SEEDS,SUNFLOWER/SESAME      FAT & INCOMPLETE PROTEIN
 2.00  1 TSP, 1 PAT     BUTTER                      HIGH FAT CONTENT
 3.00  1 TBSP,1 PKG     COFFEE CREAMER              HIGH CALORIC CONTENT
 1.00  1 TBSP           MAYONNAISE                  HIGH FAT CONTENT
 2.00  1 CUP            COFFEE
 1.00  1 CUP            TEA
 3.00  1 TSP            SUGAR,WHITE/BROWN           HIGH SUGAR CONTENT
 1.00  2                COOKIE,SUGAR,ASSORTED       HIGH CALORIC CONTENT
-------------------------------------------------------------------------------

PLEASE NOTE: THE ABOVE COMMENTS REFER TO EACH FOOD IN A GENERAL SENSE.
IF YOUR DIET IS IN NEED OF SOME CHANGE, THESE COMMENTS SHOULD HELP YOU
TO DECIDE WHICH FOODS TO AVOID - IF YOUR DIET IS FINE, CONSIDER THEM AS
'INFORMATION ONLY'.

A REMINDER: THE ANALYSIS PRESENTED ABOVE IS BASED ON STANDARDS FOR
NORMALLY HEALTHY CANADIAN CHILDREN AND ADULTS. IF YOU ARE WORRIED ABOUT
YOUR DIET WE RECOMMEND THAT YOU SEEK HELP FROM A PROFESSIONAL DIETITIAN-
NUTRITIONIST, YOUR LOCAL HEALTH DEPARTMENT OR YOUR PHYSICIAN.

WE HAVE TRIED TO INDICATE THE CHARACTERISTICS OF YOUR DIET YOU MIGHT LIKE
TO CONSIDER ADJUSTING SO THAT YOUR BODY CAN FUNCTION AT ITS BEST. WE WISH
YOU GOOD HEALTH.

COMPUTER PROGRAM DEVELOPED FOR ACTION B.C.

NUTRIENT ANALYSIS BASED ON CURRENT CANADIAN DIETARY STANDARDS.
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APPENDIX I

ACTIVITY ASSESSMENT FORM

WEEKLY ACTIVITY ASSESSMENT

Name: ______________________________ Date: ______________________

Activity Period: __________________ to _________________________

ACTIVITY	# SESSIONS	TOTAL TIME	INTENSITY (lo,med, high)
TENNIS			
SQUASH			
RACQUETBALL			
HANDBALL			
BADMINTON			
RUGBY			
BASKETBALL			
VOLLEYBALL			
TEAM HANDBALL			
ICE HOCKEY			
SOCCER			
FOOTBALL			
BASEBALL			
GOLF			
JOGGING			
SPRINTING			
STAIR RUNNING			
CYCLING			
SWIMMING			
ORIENTEERING			
X-COUNTRY SKIING			
WEIGHTLIFTING			
DOWNHILL SKIING			
OTHER:			

APPENDIX J

PROCEDURE FOR SEPARATION OF HDL-CHOLESTEROL AND (LDL+VLDL)-CHOLESTEROL

SAMPLE:

0.5 ml serum after an overnight fast of 12 hours minimum (as little as 0.2 ml may be used). Sera may be stored at 4°C. for up to one week prior to analysis or frozen up to one month. Dilute lipemic samples 1 in 2 with 0.15 M NaCl.

SEPARATION OF HDL AND LDL + VLDL:

A serum pool is run with each batch of samples.

1. Transfer 500 ml serum to a glass test tube (100 x 12 mm) using a volumetric pipette. (Smaller volumes of serum may be used with proportionate amounts of heparin and $MnCl_2$).
2. Add 25 µl heparin (5000 units/ml) using an SMI pipette. Mix well.
3. Then add 25 µl of 1 M $MnCl_2$ using an SMI pipette. Mix immediately in a vortex mixer.
4. Let the prepared samples stand at room temperature for 30 minutes.
5. Then centrifuge at 2600 rpm for 10 minutes (800 x g.).
6. Prepare a reagent blank containing 0.5 ml deionized H_2O, 25 ul heparin, and 25 µl $MnCl_2$.
7. Carefully transfer the clear supernatant HDL solution to a clean tube. If the supernatant is not clear as can occur when a sample has an increased triglyceride level, (a) repeat the precipitation with a 1 in 2 dilution of serum with 0.15 M NaCl or (b) add 0.5 ml 0.15 M NaCl, 25 µl heparin and 25 µl $MnCl_2$ to the originally prepared solution, mix well, let stand a further 10 minutes then

PRINCIPLE:

Heparin and manganese cholride ($MnCl_2$) are added directly to serum to precipitate lipoproteins of density less than 1.063 (LDL and VLDL lipoproteins) leaving HDL in solution. After 30 minutes the mixture is centrifuged, the clear supernatant (containing HDL) removed, and the precipitate dissolved in sodium citrate. Cholesterol levels are measured enzymatically in both supernatant (HDL) and dissolved precipitate (LDL + VLDL).

REAGENTS:

1.	Heparin, 5000 μ/ml	
	Dilute 1.0 ml heparin, sodium (Organon Canada Ltd. or Harris Labs).	
	(10,000 units/ml) with 0.15M NaCl	1.0 ml
2.	Sodium Chloride, (NaCl) 0.15 M	
	NaCl	0.877 g
	Dissolved in deionized H_2O and q.s. to	100 ml
3.	Manganous Chloride 1 M (F.W. 197.9)	
	$MnCl_2$, H_2O	19.79 g
	Dissolved in deionized water and q.s. to	100 ml
4.	Sodium Citrate 0.1 M	
	Sodium Citrate	2.94 g
	Dissolved in deionized water and q.s. to	100 ml

centrifuge. The supernatant should be clear.

8. Dissolve the precipitated LDL + VLDL by adding 0.5 ml sodium citrate (0.1 M). Mix well and let stand at least 10 minutes prior to analysis.

APPENDIX K

PROCEDURE FOR DETERMINATION OF SERUM LIPIDS AND LIPOPROTEINS

SERUM CHOLESTEROL - ABA 100

PRINCIPLE:

Cholesterol esters in serum are hydrolyzed to free cholesterol by cholesterol esterase

Cholesterol esters $\xrightarrow{\text{cholesterol esterase}}$ cholesterol + fatty acids

The free cholesterol produced is oxidized by cholesterol oxidase to cholest-4-en-3-one with simultaneous production of hydrogen peroxide.

Free cholesterol + O_2 $\xrightarrow{\text{cholesterol oxidase}}$ cholest-4-en-3-one + H_2O_2

Hydrogen peroxide couples oxidatively with 4-aminophenazone and phenol in the presence of peroxidase to yield a quinoneamine dye with an absorption maximum at 500 nm.

H_2O_2 + Phenol + 4-aminophenazone $\xrightarrow{\text{POD}}$ red dye + $2H_2O$

The intensity of color formed is proportional to the cholesterol concentration and can be measured photometrically between 400 and 560 nm.

INSTRUMENT PARAMETERS:

Filter	500/600
Power	ON
Incubator	30°C
Mode	END POINT
Reaction Direction	UP
Analysis time	10 mins.
Carousel revolution	2
Stringe plate	1:101
Sample size	5 μl
Decimal setting	0000
Zero	0000

REAGENTS:

1. Bio-Dynamics/BMC cholesterol CHOD-PAP Enzymatic method. Cat. # 15737.

2. Standard: Precilip BMC Cat. # 15938.

Working Reagent

Bottle (1) (Buffer/4-aminophenazone) 65 mls.
Bottle (2) (Chol.esterase/Peroxidase) 1.0 mls.

Bottle (3) (Chol.oxidase)	1.0 mls.
Bottle (4) (Phenol)	1.25 mls.
Deionized H_2O	65 mls.

Store in an amber bottle at +4°C.
This reagent is stable for one week at room temperature or four weeks at +4°C.

CAROUSEL FORMAT: (Cholesterol and Triglycerides)

Cup 01	H_2O
Cup 02	H_2O
03-05	Precilip
06-10	Serum samples
11	C8
12-20	Serum samples
*See note 2 21	Validate-E-L
22-31	Serum samples

Aliquot 50 µl of H_2O, serum samples and controls into sample cups. "Sera-seal" each cup immediately after aliquotting. The carousel is used for cholesterol and triglyceride determinations.

PROCEDURE:

1. With "POWER" OFF, position 500/600 filter. Switch POWER ON. Set operating parameters.

2. Pour sufficient working reagent for the days samples into amber reagent vials. Prime 1:101 syringe plate.

3. Load the first carousel, check the position of the sample probe in the sample cup and cuvette as described in the ABA 100 manual.

4. Start the run by pressing "STOP" then "RUN".

5. At the end of the first revolution, when the carousel is in the 00 position press "STOP" then "TEST". Remove the sample probe from the sample arm and place in the reagent vial.

6. Manually rotate the carousel to position 01. Set zero to 0000 on the display using the zero control. Rotate to position 02, check zero.

7. Rotate to position 03. Using scaling vernier adjust the nixie display to the package insert value for Precilip. Push calibrate control and record calibration factor.

8. Repeat step 7 for cup positions 04 and 05. Average the calibration factors found.

9. Push calibrate control and use the scaling vernier to adjust the display to read the averaged factor from step 8.

10. Rotate the carousel to 00 position. Push "STOP" then "RUN". Allow the carousel to move to position 01 then move the carousel on to position 00. Allow to print.

NOTES:

1. Results are printed out in mg/100 ml cholesterol.

2. Validate -E-L (Warner-Chilcott) can be used as a high control. This control serum does not store well once reconstituted. Make up one vial of Validate and run for two days whenever new working reagent is prepared.

3. Dilute specimens with high values with 0.9% Saline.

LINEARITY:

Accepted to 500 mg/100 ml.

NORMAL RANGE:

75-250 mg/100 ml.

SERUM TRIGLYCERIDES - ABA 100

PRINCIPLE:

A mixture of lipase and an esterase split the triglycerides quantitatively into fatty acids and free glycerol. The glycerol liberated will react as follows:

$$\text{Glycerol} + \text{ATP} \xleftrightarrow{\text{G.K.}} \text{glycerol-3-phosphate} + \text{ATP}$$

$$\text{ADP} + \text{PEP} \xleftrightarrow{\text{P.K.}} \text{ATP} + \text{Pyruvate}$$

$$\text{Pyruvate} + \text{NADH} + \text{H}^+ \xleftrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}^+$$

The amount of NADH oxidized during the reaction is equivalent to the amount of glycerol in the specimen. The resulting decrease in absorbance is measured photometrically at 340 nm.

INSTRUMENT PARAMETERS:

Filter	340/380 (ABA 2 FF = 4.73)
Power	ON
Incubation	37°C
Mode	END POINT
Reaction Direction	DOWN
Analysis Time	10 mins.
Carousel Revolution	2
Syringe Plate	1:51
Sample Size	5 µl
Decimal Position	0000
Zero	0000
*Calibration Factor	486

*NB Use filter with F.F. = 4.73 only.

REAGENTS:

1. Bio-Dynamics/BMC Triglycerides (Fully enzymatic)
   Cat.# 15970.

2. Bocine Albumin powder Fraction V. Cat.# 7110-05.
   Metrix Clinical and Diagnostic Division
   Armour Pharmaceutical Co.
   Chicago, Illinois 60690

3. Glycerokinase Sigma Chemical Co. Cat.# G5751

4. Standard The procedure is standardized on the extinction coefficient of NADH (see Note 3.).

Reagent Preparation

1. As new kits are received 250 mg Bocine Albumin is added to each Bottle (1) (Buffer). Mix thoroughly until dissolved.

2. Reconstitute one Bottle (2) (NADH/ATP/PEP) with 2.0 ml deionized water. (Stable for 2 weeks at $+4^{\circ}$C.) Bottles (1), (3), and (4) are used undiluted.

3. The working reagent is prepared immediately before each carousel is sampled. For one full carousel prepare the following volume of reagent.

Bottle	(1)	(Buffer)	10 mls
Bottle	(2)	(NADH/ATP/PEP)	200 μl
Bottle	(3)	(LDH/PK/Lipase/Esterase)	200 μl
Bottle	(4)	(Glycerokinase)	60 μl

Sigma glycerokinase may be used to supplement and extend the life of the BMC kit.
Half-volumes of the above reagent can be prepared as required.

PROCEDURE:

1. The carousel prepared for the cholesterol determinations is re-sampled. If additional sera are to be run the same carousel format as for cholesterols should be observed.

2. Load the 1:51 syringe plate with working reagent.

3. As soon as the cholesterol run has printed Switch Power - OFF, change the filter, Turn Power - ON and set operating parameters.

4. Place the first carousel to be run in position with a new cuvette in place. Turn to 00 carousel position.

5. In "TEST" mode set zero to 0000 against air (00 carousel position).

6. Push "CALIBRATE" button. Use "SCALING VERNIER" to enter 486 on the display.

7. Check the position of the sample probe in the sample cup and cuvette. Adjust as per ABA 100 manual if necessary.

8. Push "STOP" then "RUN". Allow to dispense and print out.

NOTES:

1. The first reagent blank (Position 01) should read in the range 9100-9300. The second reagent blank (Position 02) should read 0000 ± 4.

2. Check the results for the controls
   a) Precilip - label claim value
   b) C8

3. Standardization:
   The calculation for the triglyceride calibration factor is as follows:

$$\frac{\text{Scaling factor}}{\text{Filter factor}} \times \frac{\text{Total vol. (mls)}}{\text{Spec. vol. (mls)}} \times \frac{\text{mol. wt.}}{10} = \text{calibration factor}$$

$$\frac{0.500}{4.73} \times \frac{0.255}{0.005} \times \frac{885}{10} = 486$$

REPORT:

Serum Triglycerides = ... mg/100 ml.

NORMAL RANGE:

60-165 mg/100 ml.

LINEARITY:

Accepted to 500 mg/100 ml.
Dilute high specimens with 0.9% saline.

HDL CHOLESTEROL

Subtract reagent blank values from HDL values, and multiply corrected HDL values by 1.1 (to correct for the dilution with the addition of heparin and $MnCl_2$). (Not required for (LDL + VLDL values.)

If the sample was diluted prior to precipitation (7a), multiply both HDL and (LDL + VLDL) values by the dilution factor.

However, if the sample was diluted by adding NaCl, heparin, $MnCl_2$ to the already precipitated sample, multiply ONLY the HDL value by the dilution factor (7b).

Results are in mg/dl.

e.g. Reagent Blank = 1 mg/dl
HDL = 30 mg/dl
Then 30 - 1 = 29 mg/dl
and 29 x 1.1 = 32 mg/dl HDL Cholesterol
If the sample was diluted 1 in 2, then 32 x 2 = 64 mg/dl HDL cholesterol present.

(LDL + VLDL) CHOLESTEROL

Do not subtract reagent blank values nor apply a 1.1 correction factor.

If the sample was initially diluted (7a) and 0.5 ml used for precipitation, multiply the result by 2.

If the sample has been diluted by adding NaCl, heparin, $MnCl_2$ to the already precipitated sample, no correction factor is required since the precipitation of LDL + VLDL is dissolved in 0.5 ml citrate. See Note 6.

If no dilution correction is required, the results from the ABA-100 (in mg/dl) are taken directly for LDL + VLDL cholesterol and the native cholesterol.

NOTES:

1. Either the 1:101 or 1:51 syringe plate may be used for cholesterol estimations with good results for native serum, HDL and (LDL + VLDL) fractions. The calibration factor with the 1:51 plate will be about 180 with precilip set at 124 mg/dl.

2. EDTA plasma may be used instead of serum. However, HDL values will be about 5 mg/dl higher in plasma than in comparable serum.

3. HDL supernates must be clear after centrifugation. Any turbidity indicates incompleteness of precipitation and samples must be diluted and re-precipitated.

4. Lipoprotein electrophoresis may be performed on native serum to determine the presence of lipoprotein abnormalities, and on the HDL supernatant to verify completeness of LDL + VLDL precipitation. The method used is that performed in the routine lab. (1 μl sample on 1% agarose for 30 minutes at 20 MA, in EDTA barbital buffer).

5. A fine precipitate develops in the HDL supernatant on standing, or if left overnight at 4^{o}. This is manganese oxide, which does not interfere with cholesterol estimations.

6. If less than 0.5 ml of serum is precipitated and the precipitate dissolved in 0.5 ml sodium citrate, multiply the VLDL + LDL result by $\frac{0.5}{\text{ml of serum}}$

7. The $MnCl_2$ and heparin have been shown to have no effect on the enzyme activity for the cholesterol estimation.

APPENDIX L

STANDARD SERUM ANALYSIS:
RELIABILITY OF HDL-CHOLESTEROL DETERMINATION

RELIABILITY OF HDL-CHOLESTEROL DETERMINATION

Q_4 Serum	HDL-cholesterol
STANDARD REFERENCE	43.0 ± 3.3 ($\bar{x} \pm$ SD)
VALUES OBTAINED IN LAB:	45.2 ± 5.4
April 9	47
10	45
10	46
15	39
16	40
18	44
25	40
29	45
30	37
May 1	39
7	48
8	53
21	48
23	47
26	48
28	41
29	37
June 2	48
5	42
11	48
12	59
16	51
23	48

APPENDIX M.

WORK COMPLETED DURING EACH TRAINING SESSION

Stage I - 0 - 3 Weeks

Stage II - 3 - 6 Weeks

Stage III - 6 - 9 Weeks

WORK COMPLETED DURING EACH TRAINING SESSION (KPM)

STAGE I

	Subjects												
Mean	06	05	04	12	03	11	10	02	09	08	07	01	Training Session
35002	37800	34110	25830	39330	37530	32400	41400	40410	26280	31320	34110	39510	1
36750	38700	41490	29700	38340	39870	29700	39600	44370	29160	30510	40950	38610	2
38333	39240	43650	29700	39150	43380	33030	40050	40050	32040	36000	40050	43650	3
39622	40680	42390	35640	42840	42210	33930	41940	44010	32670	37260	41490	40410	4
39840	39330	40050	34920	42480	43110	36450	38520	44640	32310	37890	44550	43830	5
40800	40770	43470	33570	43200	42750	36900	43200	46800	32400	36990	46350	43200	6
41010	42030	40860	35280	42840	45180	35910	42030	46170	32400	37620	47250	44550	7
41377	41580	40950	35460	45540	45990	36450	43200	44460	33570	38250	47700	43380	8
40080	41580	43830	34920	42390	44640	36630	43200	30060	33030	38340	47340	45000	9
41400	40500	45900	36540	43020	42210	35280	43200	45540	32580	39510	47700	44820	10
40605	42120	41760	35100	43380	44010	35550	43200	41400	30060	38430	46800	45450	11
40995	40320	42300	36900	44550	43290	36900	43200	45360	25470	39600	48600	45450	12

x = bicycle test substituted ✓ = trained on own

- = missed session

STAGE II

	Subjects												
Mean	06	05	04	12	03	11	10	02	09	08	07	01	Training Session
	x	x	x	x	x	x	x	x	x	x	x	x	13
42099	43260	46530	36900	x	44280	38970	45540	x	33120	39600	48600	44190	14
43052	43200	45000	38700	45900	45900	39150	45900	42120	38610	40500	48600	✓	15
43527	44100	✓	40500	47520	47700	33300	45900	49950	34650	40500	49500	45180	16
44110	45900	46725	39600	46800	46350	38610	48600	49950	34380	40050	49410	42945	17
44347	44100	48600	42300	47340	44109	38250	48600	51120	33750	40050	49770	44100	18
44779	44550	46800	42570	48600	46800	39150	48600	50850	35550	40050	51300	42525	19
44280	42750	44550	39600	46530	45900	40680	48600	51840	34830	40500	51480	44100	20
43366	45450	46350	39600	47700	45900	40500	44100	✓	35640	40500	52020	39270	21
44394	45000	48600	39330	47520	45900	40590	48600	✓	36000	40500	52200	44100	22
45336	44550	50400	41400	48060	46800	39330	47250	52200	36000	40500	52200	✓	23
44912	45000	46170	40950	47700	47250	39600	48600	52200	36000	40500	51300	43680	24

x = bicycle test substituted   ✓ = trained on own

- = missed session

STAGE III

	Subjects												
Mean	06	05	04	12	03	11	10	02	09	08	07	01	Training Session
	x	x	x	x	x	x	x	x	x	x	x	x	25
45472	43200	x	x	49140	46350	40770	x	53100	36720	40500	54000	x	26
45674	44100	48600	42300	47250	46350	40500	49050	53100	35100	40050	54000	47700	27
45106	45900	48600	45720	46800	44100	39150	49500	53100	35100	40950	✓	47250	28
45368	45450	49050	45450	46800	46800	37800	49050	53100	36000	41850	✓	47700	29
45804	44550	51300	44550	46350	47070	41877	49500	53100	35100	42300	-	48150	30
45981	44550	46800	45450	45450	✓	40950	49050	53100	36000	41850	54000	48600	31
46972	45900	50490	46080	45900	48150	41850	50850	52650	36000	43200	54900	47700	32
47790	45450	50400	54000	46530	48600	40950	50400	54000	35100	42750	55800	49500	33
46950	45900	52200	46350	45450	46800	40950	49050	53100	35100	43200	56700	48600	34
46207	47250	41400	46170	46170	47700	40950	49950	53100	35100	43200	56700	46800	35
46890	45000	49950	45450	46350	-	42750	-	53550	36450	43200	56700	49500	36

x = bicycle test substituted ✓ = trained on own

- = missed session

APPENDIX N

DESIGNATION OF 'HI-FIT' AND 'LOW-FIT' GROUPS: PREDICTED $\dot{V}O_2$ MAX

PREDICTED $\dot{V}O_2$ MAX (ml/kg.min^{-1}) AT THE PRE-TEST

	EXERCISE GROUP		CONTROL GROUP	
HI FIT:	Subject #	Value	Subject #	Value
	02	57.9	14	56.7
	03	57.4	16	55.9
	06	55.8	17	54.0
	01	54.1	13	53.5
	05	53.1	15	52.5
	04	49.6		
	$\bar{x}$ =	54.7	$\bar{x}$ =	54.5
LO FIT:				
	10	48.9	21	50.3
	12	48.0	19	49.2
	11	46.7	18	42.4
	09	46.2	20	38.1
	08	35.2		
	07	35.0		
	$\bar{x}$ =	43.3	$\bar{x}$ =	45.0
Overall	$\bar{x}$ =	49.0	=	50.3

APPENDIX O

ANOVA TABLES

APPENDIX O-I

SUMMARY TABLE OF F RATIOS OBTAINED FROM THE 3 WAY ANOVA

PARAMETER	A=FITNESS	B=GROUP	AB	C=TIME	AC	BC	ABC
$\dot{V}O_2$ max (l/min^{-1})	0.274	5.035*	0.948	11.953*	0.358	2.777*	0.510
$\dot{V}O_2$ max $(ml/kg.min^{-1})$	0.317	4.264	2.577	12.778*	0.554	3.395*	0.434
HR MAX (BPM)	25.721*	0.471	0.243	4.023*	0.460	1.841	0.927
VE MAX (l/min)	1.225	13.909*	3.062	6.001*	0.210	1.222	0.468
HR 117.6 (BPM)	10.696*	3.451	0.025	1.284	0.574	3.905*	1.003
HR 176.5 (BPM)	6.477*	2.645	0.208	4.013*	0.871	2.098	0.546
WL VO_2 max	0.113	4.703*	1.444	3.452*	1.236	1.530	1.718
VO_2 117.6 (l/min^{-1})	0.003	0.554	1.183	7.276*	0.981	0.513	0.970
AT-VO_2	4.360	6.099*	0.006	0.871	2.816*	1.821	0.633
AT-PO	0.316	1.328	1.507	4.876*	2.098	5.938*	9.833
AT ml	0.282	0.216	1.368	8.027*	1.542	4.651*	0.719
serum cholesterol (mg/100 ml)	0.080	0.046	0.535	1.690	0.520	2.117	1.221

*F Ratio significant at 0.05 level

SUMMARY TABLE OF F RATIOS OBTAINED FROM THE 3 WAY ANOVA CONTINUED

PARAMETER	A=FITNESS	B=GROUP	AB	C=TIME	AC	BC	ABC
HDL-cholesterol (mg/100ml)	1.475	1.506	2.759	4.390*	1.966	1.011	1.470
(VLDL+LDL)-cholesterol (mg/100ml)	0.350	0.586	0.029	3.456*	0.115	1.906	0.585
HDL/TC	0.611	1.107	0.998	4.182*	0.867	1.236	0.632
serum triglyceride (mg/100 ml)	3.237	0.759	0.232	1.630	0.468	0.889	0.522
weight (kg)	0.019	0.021	0.347	4.842*	1.277	2.052	0.602
% protein	0.001	0.009	0.005	5.313*	1.534	1.812	0.000
% carbohydrate	0.527	5.688*	0.062	6.127*	0.055	0.046	0.413
% fat	0.002	2.516	0.006	0.149	1.835	0.127	1.916
caloric intake	0.528	0.060	0.046	0.039	0.029	2.117	0.036
% body fat	0.291	3.039	0.419	29.944*	1.892	3.011	1.181

*F Ratio significant at 0.05 level

APPENDIX O-II

SUMMARY TABLE OF F RATIOS OBTAINED FROM THE 2 WAY ANOVA

PARAMETER	A=GROUP	B=TIME	AB
% ST Fibers	0.205	1.542	0.610
% FTa Fibers	2.503	2.484	1.549
% FTb Fibers	0.162	0.510	0.033
SDH activity (moles x g x min^{-1})	1.117	2.245	0.247

*** F Ratio significant at 0.05 level**

APPENDIX P

CORRELATION OF METABOLIC AND LOCAL MUSCLE PARAMETERS

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- - - - - - - - - - - - P E A R S O N   C O R R E L A T I O N   C O E F F I C I E N T S - - - - - - - - - - - -

             ST          MVO2        ATOP        ATML        ATV         HR

ST           1.0000      0.1238      0.2398      0.2253      0.2286      0.1006
            (    0)     (   11)     (   11)     (   11)     (   11)     (   11)
            P=*****     P=0.358     P=0.239     P=0.253     P=0.249     P=0.384

MVO2         0.1238      1.0000      0.2435      0.6196      0.1985     -0.4623
            (   11)     (    0)     (   11)     (   11)     (   11)     (   11)
            P=0.358     P=*****     P=0.235     P=0.021     P=0.279     P=0.076

ATOP         0.2398      0.2435      1.0000      0.7140      0.7279      0.2485
            (   11)     (   11)     (    0)     (   11)     (   11)     (   11)
            P=0.239     P=0.235     P=*****     P=0.007     P=0.006     P=0.231

ATML         0.2253      0.6196      0.7140      1.0000      0.8884      0.1666
            (   11)     (   11)     (   11)     (    0)     (   11)     (   11)
            P=0.253     P=0.021     P=0.007     P=*****     P=0.000     P=0.312

ATV          0.2286      0.1985      0.7279      0.8884      1.0000      0.4610
            (   11)     (   11)     (   11)     (   11)     (    0)     (   11)
            P=0.249     P=0.279     P=0.006     P=0.000     P=*****     P=0.077

HR           0.1006     -0.4623      0.2485      0.1666      0.4610      1.0000
            (   11)     (   11)     (   11)     (   11)     (   11)     (    0)
            P=0.384     P=0.076     P=0.231     P=0.312     P=0.077     P=*****

(COEFFICIENT / (CASES) / SIGNIFICANCE)      (A VALUE OF 99.0000 IS PRINTED IF A COEFFICIENT CANNOT BE COMPUTED)
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- - - - - - - - - - - - P E A R S O N   C O R R E L A T I O N   C O E F F I C I E N T S - - - - - - - - - - - -

             SDH         ST          HR          ATV         ATML        ATPO        MVO2

SDH          1.0000     -0.3280      0.1224     -0.1083      0.5636     -0.3298     -0.1988
            (    0)     (    7)     (    5)     (    5)     (    5)     (    5)     (    8)
            P=*****     P=0.236     P=0.422     P=0.431     P=0.161     P=0.294     P=0.318

ST          -0.3280      1.0000     -0.3279     -0.6164     -0.7832      0.7775     -0.2828
            (    7)     (    0)     (    5)     (    5)     (    5)     (    5)     (    7)
            P=0.236     P=*****     P=0.295     P=0.134     P=0.059     P=0.061     P=0.269

HR           0.1224     -0.3279      1.0000      0.8462      0.1171      0.2786     -0.5943
            (    5)     (    5)     (    0)     (    5)     (    5)     (    5)     (    5)
            P=0.422     P=0.295     P=*****     P=0.035     P=0.426     P=0.325     P=0.145

ATV         -0.1083     -0.6164      0.8462      1.0000      0.1830     -0.1187     -0.6505
            (    5)     (    5)     (    5)     (    0)     (    5)     (    5)     (    5)
            P=0.431     P=0.134     P=0.035     P=*****     P=0.384     P=0.425     P=0.117

ATML         0.5636     -0.7832      0.1171      0.1830      1.0000     -0.5717      0.6255
            (    5)     (    5)     (    5)     (    5)     (    0)     (    5)     (    5)
            P=0.161     P=0.059     P=0.426     P=0.384     P=*****     P=0.157     P=0.130

ATPO        -0.3298      0.7775      0.2786     -0.1187     -0.5717      1.0000     -0.3423
            (    5)     (    5)     (    5)     (    5)     (    5)     (    0)     (    5)
            P=0.294     P=0.061     P=0.325     P=0.425     P=0.157     P=*****     P=0.286

MVO2        -0.1988     -0.2828     -0.5943     -0.6505      0.6255     -0.3423      1.0000
            (    8)     (    7)     (    5)     (    5)     (    5)     (    5)     (    0)
            P=0.318     P=0.269     P=0.145     P=0.117     P=0.130     P=0.286     P=*****

(COEFFICIENT / (CASES) / SIGNIFICANCE)      (A VALUE OF 99.0000 IS PRINTED IF A COEFFICIENT CANNOT BE COMPUTED)
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APPENDIX Q

RAW DATA

APPENDIX Q

RAW DATA

APPENDIX I	Serum Lipids and Lipoporteins
APPENDIX II	Bicycle Ergometer Test
APPENDIX III	Anaerobic Threshold
APPENDIX IV	Body Composition and Diet
APPENDIX V	SDH Activity
APPENDIX VI	Muscle Fiber Types

Q-1 Serum Lipids

COLUMNS: 1,2 Subject ID

4 Fitness (1 = Hi-fit 2 = Lo-fit)

6 Group (1 = Exercise 2 = Control)

8 Variable Block

10 Variable:
1 = serum cholesterol
2 = serum HDL-cholesterol
3 = (VLDL + LDL)-cholesterol
4 = HDL-cholesterol/Total cholesterol
5 = serum triglyceride

(-1 = missing data)

```
01 1 1 1 1 185 191 194 189 200 185 186
01 1 1 1 2 57 61 68 62 68 73 59
01 1 1 1 3 128 130 126 127 132 112 127
01 1 1 1 4 .31 .32 .35 .33 .34 .41 .32
01 1 1 1 5 077 065 096 062 053 058 084
02 1 1 1 1 277 248 232 290 274 267 228
02 1 1 1 2 74 63 66 86 75 -1 79
02 1 1 1 3 203 185 166 204 199  -1 149
02 1 1 1 4 .27 .25 .29 .30 .27  -1 .35
02 1 1 1 5 142 129 123 118 155 102 091
03 1 1 1 1 155 171 174 177 192 174 189
03 1 1 1 2 62 50 77 66 70 70 66
03 1 1 1 3 093 121 097 111 122 104 123
03 1 1 1 4 .40 .29 .44 .37 .37 .40 .35
03 1 1 1 5 075 064 062 053 044 066 040
04 1 1 1 1 171 197 172 173 190 155 172
04 1 1 1 2 60 44 54 51 62 59 57
04 1 1 1 3 111 153 118 122 128 096 115
04 1 1 1 4 .35 .22 .31 .30 .33 .38 .33
04 1 1 1 5 088 078 063 059 078 076 061
05 1 1 1 1 186 203 171 187 195 188 205
05 1 1 1 2 59 66 59 59 68 75 66
05 1 1 1 3 127 137 112 128 127 113 139
05 1 1 1 4 .32 .33 .35 .32 .35 .40 .32
05 1 1 1 5 084 074 113 054 064 051 139
06 1 1 1 1 140 135 138 133 134 140 135
06 1 1 1 2 47 61 48 45 48 51 48
06 1 1 1 3 093 074 090 088 086 089 090
06 1 1 1 4 .34 .45 .35 .34 .36 .36 .35
06 1 1 1 5 078 091 066 051 078 066 068
07 2 1 1 1 195 182 188 179 196 156 174
07 2 1 1 2 38 37 49 57 42 -1 51
07 2 1 1 3 157 145 139 122 154  -1 123
07 2 1 1 4 .20 .21 .26 .32 .21  -1 .29
07 2 1 1 5 178 165 101 088 137 078 101
08 2 1 1 1 223 211 215 178 207 199 256
08 2 1 1 2 48 40 48 42 46 53 48
08 2 1 1 3 175 171 167 136 161 146 208
08 2 1 1 4 .22 .19 .23 .24 .22 .27 .19
08 2 1 1 5 158 108 140 078 136 143 137
09 2 1 1 1 133 143 139 140 150 148 148
09 2 1 1 2 35 44 48 53 57 -1 44
09 2 1 1 3 098 099 091 087 093  -1 104
09 2 1 1 4 .26 .31 .35 .38 .38  -1 .30
09 2 1 1 5 175 094 117 125 092 099 161
10 2 1 1 1 161 163 159 165 169 157 167
10 2 1 1 2 53 66 55 54 66 68 59
10 2 1 1 3 108 097 104 111 103 089 108
10 2 1 1 4 .33 .41 .35 .33 .39 .43 .35
10 2 1 1 5 115 080 095 079 121 103 058
11 2 1 1 1 162 150 162 145 176 155 147
11 2 1 1 2 55 46 46 43 51 53 48
11 2 1 1 3 107 104 116 102 125 102 099
11 2 1 1 4 .34 .31 .29 .30 .29 .34 .33
11 2 1 1 5 072 072 079 060 072 055 135
12 2 1 1 1 184 193 165 164 189 167 164
12 2 1 1 2 43 54 59 45 55 48 53
12 2 1 1 3 141 139 106 120 134 119 111
12 2 1 1 4 .23 .28 .36 .27 .29 .29 .32
12 2 1 1 5 122 078 096 090 130 105 077
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13 1 2 1 1 196 202 212 192 163 168 173
13 1 2 1 2 53 42 44 49 53 69 53
13 1 2 1 3 143 160 168 143 110 100 120
13 1 2 1 4 .27 .21 .21 .26 .33 .41 .31
13 1 2 1 5 107 134 070 084 097 074 076
14 1 2 1 1 168 161 189 168 180 175 186
14 1 2 1 2 51 52 66 48 51 59 55
14 1 2 1 3 117 109 123 120 129 116 131
14 1 2 1 4 .30 .32 .35 .29 .28 .34 .30
14 1 2 1 5 092 125 129 077 103 079 058
15 1 2 1 1 167  -1 151 146 152 146 144
15 1 2 1 2 55 -1 53 53 55 51 55
15 1 2 1 3 112  -1 098 093 097 095 089
15 1 2 1 4 .33  -1 .35 .36 .36 .35 .38
15 1 2 1 5 069  -1 069 044 052 058 065
16 1 2 1 1 175 173 177  -1 221  -1  -1
16 1 2 1 2 46 47 46 -1 62 -1 -1
16 1 2 1 3 129 126 131  -1 159  -1  -1
16 1 2 1 4 .26 .27 .26  -1 .28  -1  -1
16 1 2 1 5 113 087 079  -1 094  -1  -1
17 1 2 1 1 193 176 177 160 170 195 184
17 1 2 1 2 51 40 46 53 48 55 46
17 1 2 1 3 142 136 131 107 122 130 129
17 1 2 1 4 .26 .23 .26 .33 .28 .30 .30
17 1 2 1 5 073 058 074 057 061 058 156
18 2 2 1 1 234 264 257 259 242 233 244
18 2 2 1 2 46 42 44 51 57 46 48
18 2 2 1 3 188 222 213 208 185 187 195
18 2 2 1 4 .20 .16 .17 .20 .24 .20 .20
18 2 2 1 5 226 273 217 245 287 310 324
19 2 2 1 1 180 199 205 170 200 168 176
19 2 2 1 2 48 51 40 54 62 48 51
19 2 2 1 3 132 148 165 116 138 120 125
19 2 2 1 4 .27 .25 .19 .32 .31 .29 .29
19 2 2 1 5 101 076 095 053 071 085 052
20 2 2 1 1 160 169 185 176 158 154 154
20 2 2 1 2 52 54 59 59 59 59 53
20 2 2 1 3 108 115 126 117 099 095 101
20 2 2 1 4 .33 .32 .32 .34 .37 .38 .34
20 2 2 1 5 120 137 106 139 091 072 112
21 2 2 1 1 153 137 152 152 152 134 170
21 2 2 1 2 57 56 57 59 66 51 51
21 2 2 1 3 096 081 095 093 086 083 119
21 2 2 1 4 .37 .41 .38 .39 .43 .38 .30
21 2 2 1 5 084 074 102 108 056 094 091
```

Q-II Bicycle Ergometer Test

COLUMNS: 1,2 Subject ID

4 Fitness (1 = Hi-fit 2 = Lo-fit)

6 Group (1 = Exercise 2 = Control)

8 Variable Block

10 Variable:
1 = Maximum heart rate
2 = VO_2 max ($ml/kg \cdot min^{-1}$)
3 = VO_2max (l/min^{-1})
4 = VE max
5 = HR 117.6
6 = HR 176.5
7 = Work load at VO_2 max
8 = VO_2 117.6
9 = VO_2 176.5

(-1 = missing data)

```
01 1 1 3 1 184 175 176 174 176 172 171
01 1 1 3 2 63.4 72.7 60.7 69.7 66.0 62.8 55.6
01 1 1 3 3 4.85 5.53 4.59 5.28 4.96 4.68 4.13
01 1 1 3 4 178.6 171.9 178.3 175.8 164.7 171.0 172.5
01 1 1 3 5 118 106 112 098 120 109 112
01 1 1 3 6 144 128 139 128 137 131 132
01 1 1 3 7 294.1 338.2 338.2 338.2 338.2 323.5 323.5
01 1 1 3 8 1.41 1.78 1.76 1.58 1.81 1.63 1.68
01 1 1 3 9 2.15 2.39 2.31 2.58 2.59 2.31 2.37
02 1 1 3 1 180 180 180 172 161  -1 169
02 1 1 3 2 50.7 55.7 58.8 54.1 54.6   -1 52.0
02 1 1 3 3 4.41 4.96 5.18 4.82 4.88   -1 4.61
02 1 1 3 4 177.3 188.0 199.9 195.9 179.8    -1 181.1
02 1 1 3 5 112 122 116 110 123  -1 116
02 1 1 3 6 130 129 130 118 135  -1 136
02 1 1 3 7 323.5 338.2 323.5 352.9 338.2    -1 294.1
02 1 1 3 8 1.65 1.60 1.73 1.81 1.59   -1 1.82
02 1 1 3 9 2.40 2.29 2.31 2.42 2.50   -1 2.78
03 1 1 3 1 178 176 175 176 180 183 182
03 1 1 3 2 52.8 63.9 68.2 71.6 59.5 66.2 63.2
03 1 1 3 3 3.73 4.45 4.76 4.98 4.13 4.63 4.43
03 1 1 3 4 137.0 161.2 174.2 177.8 158.2 164.2 153.4
03 1 1 3 5 138 113 126 108 114 134 123
03 1 1 3 6 145 142 151 138 145 158 152
03 1 1 3 7 294.1 323.5 323.5 338.2 338.2 323.5 323.5
03 1 1 3 8 1.57 1.66 1.90 1.53 1.59 1.84 1.64
03 1 1 3 9 2.19 2.49 2.73 2.50 2.20 2.51 2.34
04 1 1 3 1 195 192 186 177 178 180 177
04 1 1 3 2 46.2 57.6 54.7 72.9 62.9 65.2 64.5
04 1 1 3 3 3.19 4.06 3.90 5.15 4.53 4.68 4.55
04 1 1 3 4 147.3 152.9 162.8 165.5 168.2 163.6 156.7
04 1 1 3 5 132 124 125 110 114 121 115
04 1 1 3 6 160 147 150 137 142 147 142
04 1 1 3 7 264.7 308.8 294.1 323.5 338.2 294.1 264.7
04 1 1 3 8 1.39 1.79 1.56 1.74 1.80 1.70 1.64
04 1 1 3 9 2.14 2.42 2.02 2.44 2.63 2.43 2.37
05 1 1 3 1 184 185 186 186 182 192 180
05 1 1 3 2 47.3 57.3 50.3 56.4 55.6 49.7 50.1
05 1 1 3 3 3.60 4.46 3.93 4.30 4.32 3.85 3.94
05 1 1 3 4 151.1 150.8 178.8 156.8 154.7 157.0 138.9
05 1 1 3 5 112 112 114 115 099 118 102
05 1 1 3 6 150 134 134 135 124 136 123
05 1 1 3 7 294.1 323.5 323.5 338.2 352.9 352.9 338.2
05 1 1 3 8 1.17   -1 1.23 1.80 1.39 1.68 1.55
05 1 1 3 9 1.81 2.46 1.95 2.35 2.34 2.21 2.18
06 1 1 3 1 183 180 182 181 181 188 186
06 1 1 3 2 44.0 55.3 57.7 70.9 62.5 69.8 65.6
06 1 1 3 3 3.19 4.04 4.13 4.94 4.37 4.84 4.61
06 1 1 3 4 138.8 149.9 158.3 164.7 165.9 166.7 156.2
06 1 1 3 5 120 107 102 106 112 110 116
06 1 1 3 6 140 134 128 131 136 140 138
06 1 1 3 7 294.1 294.1 294.1 294.1 294.1 294.1 323.5
06 1 1 3 8 1.12 1.66 1.62 1.97 1.82 1.80 1.64
06 1 1 3 9 1.63 2.38 2.49 2.41 2.60 2.39 2.27
07 2 1 3 1 210 195 195 196 200 200 198
07 2 1 3 2 38.1 49.2 49.3 66.5 59.5 58.9 58.4
07 2 1 3 3 3.53 4.56 4.54 5.95 5.37 5.12 5.15
07 2 1 3 4 161.7 177.9 182.5 192.6 192.7 184.9 180.4
07 2 1 3 5 141 143 139 125 126 126 130
07 2 1 3 6 168 157 156 142 150 141 145
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07 2 1 3 7 294.1 338.2 323.5 335.2 367.6 352.9 352.9
07 2 1 3 8 1.05 1.92 1.46 1.85 1.71 1.77 1.77
07 2 1 3 9 1.38 2.67 2.10 2.51 2.51 2.62 2.51
08 2 1 3 1 194 188 185 191 192 183 187
08 2 1 3 2 33.2 37.1 37.4 41.1 42.2 34.8 35.6
08 2 1 3 3 3.31 3.79 3.72 3.90 3.99 3.23 3.48
08 2 1 3 4 145.3 145.6 143.2 147.4 145.5 127.2 139.8
08 2 1 3 5 123 124 121 129 123 122 122
08 2 1 3 6 160 146 140 158 149 151 139
08 2 1 3 7 294.1 294.1 294.1 294.1 294.1 264.7 264.7
08 2 1 3 8 1.29 1.82 1.89 1.76 1.63 1.46 1.35
08 2 1 3 9 1.97 2.55 2.50 2.32 2.25 2.21 2.12
09 2 1 3 1 195 192 190 192 196 196 189
09 2 1 3 2 38.0 51.7 58.0 63.6 63.0 52.5 63.6
09 2 1 3 3 2.20 3.05 3.44 3.62 3.61 3.08 3.66
09 2 1 3 4 122.9 131.9 157.8 153.9 145.7 155.2 143.4
09 2 1 3 5 158 141 142 145 143 136 150
09 2 1 3 6 184 180 175 171 178 171 178
09 2 1 3 7 235.3 235.3 250.0 264.7 235.3 264.7 279.4
09 2 1 3 8 1.22 1.59 1.72 1.64 1.51 1.56 1.58
09 2 1 3 9 1.69 2.38 2.68 2.39 2.35 2.16 2.16
10 2 1 3 1 185 184 182 182 190 182 174
10 2 1 3 2 51.3 59.4 59.3 66.9 63.3 56.0 61.0
10 2 1 3 3 3.96 4.57 4.43 5.05 4.65 4.09 4.46
10 2 1 3 4 174.2 178.4 184.1 191.0 182.6 168.0 148.5
10 2 1 3 5 142 128 128 128 124 131 118
10 2 1 3 6 151 146 142 141 153 155 144
10 2 1 3 7 294.1 294.1 323.5 323.5 294.1 235.3 308.8
10 2 1 3 8 1.70 1.64 1.52 2.02 1.82 1.72 1.79
10 2 1 3 9 2.25 2.56 2.07 2.63 2.39 2.44 2.30
11 2 1 3 1 195 192 192 184 192 192 189
11 2 1 3 2 40.8 62.5 63.7 70.2 60.9 59.2 60.4
11 2 1 3 3 2.52 3.79 3.96 4.30 3.75 3.69 3.72
11 2 1 3 4 141.8 153.4 162.7 164.7 157.5 156.8 135.1
11 2 1 3 5 145 121 126 127 131 137 135
11 2 1 3 6 176 155 151 151 154 162 164
11 2 1 3 7 235.3 308.8 323.5 294.1 264.7 264.7 279.4
11 2 1 3 8 1.08 1.50 1.74 1.61 1.60 1.64 1.58
11 2 1 3 9 1.59 2.31 2.50 2.22 2.26 2.51 2.23
12 2 1 3 1 192 190 195 184 191 191 189
12 2 1 3 2 47.8 58.0 56.6 56.8 57.4 56.7 52.3
12 2 1 3 3 3.94 4.71 4.49 4.52 4.59 4.43 4.20
12 2 1 3 4 137.5 150.9 145.2 162.6 144.5 146.5 146.2
12 2 1 3 5 120 109 127 108 116 120 124
12 2 1 3 6 150 135 157 133 146 159 156
12 2 1 3 7 294.1 323.5 323.5 323.5 323.5 308.8 308.8
12 2 1 3 8 1.55 1.70 1.74 1.83 1.65 1.59 1.83
12 2 1 3 9 2.54 2.33 2.40 2.55 2.40 2.27 2.46
13 1 2 3 1 180 185 188 173 180 163 162
13 1 2 3 2 45.7 44.6 44.7 42.4 41.9 40.9 44.0
13 1 2 3 3 4.04 4.10 4.12 3.94 3.76 3.58 3.83
13 1 2 3 4 130.3 151.5 151.9 128.4 155.0 127.5 140.3
13 1 2 3 5 110 112 125 122 108 111 108
13 1 2 3 6 134 142 147 142 132 136 129
13 1 2 3 7 294.1 308.8 294.1 294.1 294.1 264.7 264.7
13 1 2 3 8   -1 1.88 1.57 1.54 1.62 1.76 1.61
13 1 2 3 9   -1 2.34 2.11 2.14 2.32 2.45 2.60
14 1 2 3 1 185 186 186 194 183 182 177
14 1 2 3 2 46.1 54.8 50.0 56.3 50.3 50.9 58.7
14 1 2 3 3 3.51 4.21 3.91 4.34 3.95 4.00 4.54
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14 1 2 3 4 118.1 126.4 125.3 132.9 107.4 134.0 121.0
14 1 2 3 5 114 121 123 126 114 111 109
14 1 2 3 6 144 144 146 146 141 139 134
14 1 2 3 7 294.1 279.4 294.1 338.2 294.1 323.5 294.1
14 1 2 3 8 1.50 1.54 1.81 1.82 1.63 1.71 1.80
14 1 2 3 9 2.19 2.46 2.18 2.23 2.32 2.59 2.49
15 1 2 3 1 190   -1 180 179 173 173 177
15 1 2 3 2 40.3    -1 43.8 56.4 58.7 51.6 63.4
15 1 2 3 3 3.10    -1 3.48 4.39 4.67 4.11 5.06
15 1 2 3 4 148.9     -1 139.5 159.7 162.5 156.7 158.7
15 1 2 3 5 116   -1 111 108 106 108 114
15 1 2 3 6 148   -1 142 137 135 137 141
15 1 2 3 7 264.7     -1 264.7 294.1 294.1 294.1 308.8
15 1 2 3 8 1.10    -1 1.92 1.91 1.84 1.61 1.83
15 1 2 3 9 1.49    -1 2.21 2.57 2.57 2.41 2.51
16 1 2 3 1 190 195 196 182 192  -1  -1
16 1 2 3 2 41.9 58.4 50.1 62.6 65.3    -1    -1
16 1 2 3 3 2.97 4.08 3.31 4.10 4.42    -1    -1
16 1 2 3 4 142.3 159.3 159.4 155.5 164.4     -1     -1
16 1 2 3 5 115 135 134 117 126  -1  -1
16 1 2 3 6 145 170 164 142 154  -1  -1
16 1 2 3 7 294.1 250.0 279.4 294.1 323.5     -1     -1
16 1 2 3 8 1.32 1.66 1.43 1.74 1.67    -1    -1
16 1 2 3 9 1.88 2.64 2.14 2.44 2.37    -1    -1
17 1 2 3 1 185 180 187 182 180 188 186
17 1 2 3 2 42.3 51.9 48.5 53.1 54.8 52.1 53.7
17 1 2 3 3 2.46 3.08 2.91 3.13 3.19 3.06 3.17
17 1 2 3 4 112.7 107.8 115.9 121.2 117.7 120.4 121.4
17 1 2 3 5 126 135 139 134 148 148 154
17 1 2 3 6 170 160 178 167 172 178 177
17 1 2 3 7 235.3 205.9 205.9 235.3 235.3 205.9 205.9
17 1 2 3 8 1.16 1.58 1.71 1.62 1.71 1.66 1.66
17 1 2 3 9 1.98 2.43 2.50 2.26 2.45 2.39 2.39
18 2 2 3 1 191 192 193 187 200 193 185
18 2 2 3 2 48.3 54.5 44.2 48.1 48.1 46.0 48.0
18 2 2 3 3 3.90 4.49 3.60 3.94 3.93 3.80 3.86
18 2 2 3 4 129.4 134.4 136.2 136.0 135.4 145.6 128.3
18 2 2 3 5 127 133 138 117 136 128 121
18 2 2 3 6 160 160 157 141 158 154 143
18 2 2 3 7 294.1 294.1 294.1 323.5 294.1 323.5 323.5
18 2 2 3 8 2.20 1.92 1.60 1.61 1.87 1.68 1.59
18 2 2 3 9 2.62 2.62 2.16 2.49 2.42 2.42 2.40
19 2 2 3 1 195 195 195 195 190 192 190
19 2 2 3 2 50.3 53.4 51.5 55.3 58.0 55.7 57.1
19 2 2 3 3 3.31 3.55 3.39 3.56 3.80 3.65 3.70
19 2 2 3 4 126.5 155.1 152.8 139.1 152.1 160.3 155.7
19 2 2 3 5 136 144 141 147 141 138 133
19 2 2 3 6 165 162 161 165 158 161 154
19 2 2 3 7 264.7 308.8 264.7 294.1 294.1 279.4 308.8
19 2 2 3 8 1.49 1.78 1.38 1.84 1.58 1.60 1.62
19 2 2 3 9 2.28 2.40 2.14 2.28 2.34 2.27 2.22
20 2 2 3 1 196 198 192 195 192 196 190
20 2 2 3 2 34.8 48.8 49.7 49.0 43.2 54.3 50.4
20 2 2 3 3 2.79 4.00 4.02 4.02 3.47 4.34 4.02
20 2 2 3 4 167.1 160.4 157.7 166.2 149.2 158.3 156.8
20 2 2 3 5 149 155 135 143 148 147 145
20 2 2 3 6 174 176 160 164 170 172 166
20 2 2 3 7 205.9 235.3 264.7 264.7 235.3 279.4 264.7
20 2 2 3 8 1.40 2.13 1.54 1.79 1.69 1.62 1.88
20 2 2 3 9 2.05 2.35 2.15 2.45 2.43 2.77 2.43
```

```
21 2 2 3 1 200 200 195 196 183 192 192
21 2 2 3 2 50.5 59.2 65.2 55.5 60.5 56.9 64.9
21 2 2 3 3 3.25 3.84 4.19 3.55 3.82 3.50 4.02
21 2 2 3 4 117.2 122.6 152.0 145.3 143.6 141.8 134.3
21 2 2 3 5 135 142 142 134 126 134 135
21 2 2 3 6 165 172 173 161 146 162 160
21 2 2 3 7 294.1 264.7 264.7 264.7 294.1 294.1 294.1
21 2 2 3 8 1.46 1.73 1.84 1.63 1.63 1.70 1.59
21 2 2 3 9 2.27 2.60 2.20 2.49 2.23 2.34 2.44
```

Q-III Anaerobic Threshold

COLUMNS:	1,2	Subject ID	
	4	Fitness	(1 = Hi-fit 2 = Lo-fit)
	6	Group	(1 = Exercise 2 = Control)
	8	Variable Block	
	10	Variable:	1 = AT-VO_2 2 = AT-PO 3 = ATml

(-1 = missing data)

```
01 1 1 2 1 049.7 066.0 073.3 064.7 072.4 069.4 097.1
01 1 1 2 2 205.9 264.7 264.7 264.7 264.7 264.7 264.7
01 1 1 2 3 31.5 48.0 44.5 45.1 47.8 43.6 54.0
02 1 1 2 1 054.4 071.8 068.7 083.7 059.2    -1 060.4
02 1 1 2 2 176.5 264.7 235.3 294.1 205.9    -1 176.5
02 1 1 2 3 27.6 40.0 40.4 45.3 32.3    -1 31.4
03 1 1 2 1 056.9 055.8 075.5 073.3 053.1 054.1 067.2
03 1 1 2 2 176.5 176.5 235.3 264.7 176.5 176.5 205.9
03 1 1 2 3 31.1 35.7 51.5 52.5 31.6 35.8 42.5
04 1 1 2 1 054.3 091.3 091.2 058.2 076.5 074.8 058.8
04 1 1 2 2 147.1 235.3 264.7 264.7 235.3 235.3 205.9
04 1 1 2 3 25.1 46.8 49.9 49.7 48.2 49.5 37.9
05 1 1 2 1 057.3 065.5 072.4 084.8 060.1 074.0 063.9
05 1 1 2 2 205.9 235.3 264.7 294.1 205.9 235.3 205.9
05 1 1 2 3 27.1 37.6 36.4 47.8 33.4 35.8 32.0
06 1 1 2 1 051.1 072.3 083.0 066.7 077.9 064.0 072.9
06 1 1 2 2 176.5 235.3 264.7 235.3 264.7 235.3 264.7
06 1 1 2 3 22.5 40.0 47.9 47.3 48.7 44.7 47.8
07 2 1 2 1 059.8 080.5 077.5 071.3 085.6 052.3 049.7
07 2 1 2 2 235.3 264.7 294.1 294.1 294.1 205.9 205.9
07 2 1 2 3 22.8 39.6 38.2 47.4 49.2 30.8 29.0
08 2 1 2 1 069.6 067.4 077.5 094.2 056.6 058.9 060.7
08 2 1 2 2 205.9 176.5 205.9 264.7 176.5 147.1 176.5
08 2 1 2 3 23.1 25.0 29.0 38.7 23.9 20.5 21.6
09 2 1 2 1 076.1 077.9 080.9 074.5 064.9 070.1 059.1
09 2 1 2 2 176.5 176.5 205.9 205.9 176.5 176.5 176.5
09 2 1 2 3 28.9 40.3 46.9 47.4 40.9 36.8 37.6
10 2 1 2 1 071.3 055.9 065.6 075.3 085.6 100.0 077.2
10 2 1 2 2 205.9 176.5 235.3 264.7 264.7 235.3 235.3
10 2 1 2 3 36.6 33.2 38.9 50.4 54.2 56.0 47.1
11 2 1 2 1 100.0 079.1 082.7 080.6 080.5 083.1 081.1
11 2 1 2 2 235.3 235.3 235.3 264.7 235.3 235.3 235.3
11 2 1 2 3 40.8 49.5 52.7 56.6 49.0 49.2 49.0
12 2 1 2 1 066.3 054.8 077.6 098.4 061.0 073.2 074.6
12 2 1 2 2 205.9 205.9 264.7 294.1 205.9 235.3 205.9
12 2 1 2 3 31.7 31.8 43.9 55.9 35.0 41.5 39.0
13 1 2 2 1 077.9 063.9 067.3 054.2 095.9 086.8 079.5
13 1 2 2 2 235.3 205.9 205.9 176.5 235.3 205.9 235.3
13 1 2 2 3 35.6 28.5 30.1 23.0 40.2 35.5 35.0
14 1 2 2 1 070.7 082.8 093.4 073.9 093.3 081.7 066.1
14 1 2 2 2 205.9 235.3 235.3 235.3 235.3 235.3 235.3
14 1 2 2 3 32.6 45.4 41.7 41.6 41.9 41.6 38.8
15 1 2 2 1 055.1    -1 063.2 058.5 078.9 070.8 058.8
15 1 2 2 2 205.9    -1 176.5 176.5 235.3 205.9 205.9
15 1 2 2 3 22.2    -1 27.7 33.0 46.3 36.7 37.3
16 1 2 2 1 085.9 082.0 070.5 074.9 053.4    -1    -1
16 1 2 2 2 235.3 205.9 205.9 235.3 176.5    -1    -1
16 1 2 2 3 36.0 47.9 35.3 46.9 34.9    -1    -1
17 1 2 2 1 061.7 064.5 085.8 070.2 077.2 068.9 068.5
17 1 2 2 2 147.1 147.1 176.5 147.1 147.1 147.1 147.1
17 1 2 2 3 26.1 33.5 41.6 37.3 42.3 35.9 36.8
18 2 2 2 1 084.5 066.6 077.4 082.1 080.7 075.2 085.4
18 2 2 2 2 235.3 205.9 205.9 264.5 235.3 205.9 235.3
18 2 2 2 3 40.8 36.3 34.2 39.5 38.8 34.6 41.0
19 2 2 2 1 100.1 084.6 079.6 082.1 073.3 098.6 080.4
19 2 2 2 2 264.7 264.7 235.3 235.3 235.3 264.7 264.7
19 2 2 2 3 50.3 45.2 41.0 45.4 42.5 54.9 45.9
20 2 2 2 1 073.6 083.6 053.5 084.3 080.6 063.9 060.5
20 2 2 2 2 176.5 176.5 176.5 205.9 205.9 176.5 176.5
20 2 2 2 3 25.6 40.8 26.6 41.3 34.8 34.7 30.5
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21 2 2 2 1 090.5 082.6 071.6 089.0 083.8 086.1 080.9
21 2 2 2 2 235.3 235.3 205.9 235.3 235.3 235.3 235.3
21 2 2 2 3 45.7 48.9 46.7 49.4 50.7 49.0 52.5
```

Q-IV Body Composition and Diet

COLUMNS: 1,2 Subject ID

4 Fitness (1 = Hi-fit 2 = Lo-fit)

6 Group (1 = Exercise 2 = Control)

8 Variable Block

10 Variable: 1 = Weight
2 = % protein
3 = % carbohydrate
4 = % fat
5 = caloric intake
6 = % body fat

(-1 = missing data)

```
01 1 1 4 1 076.5 076.0 075.5 075.6 075.1 074.5 074.3
01 1 1 4 2 17.3 15.1
01 1 1 4 3 36.7 35.6
01 1 1 4 4 35.7 35.8
01 1 1 4 5 2291 2604
01 1 1 4 6 16.2 12.6 11.9
02 1 1 4 1 087.1 089.1 088.1 089.1 089.5    -1 088.7
02 1 1 4 2 16.0 13.4
02 1 1 4 3 38.8 32.4
02 1 1 4 4 38.8 32.4
02 1 1 4 5 3759 3938
02 1 1 4 6 19.0 17.9 17.3
03 1 1 4 1 070.6 069.7 069.8 069.5 069.4 069.9 070.0
03 1 1 4 2 18.9 16.4
03 1 1 4 3 54.5 43.0
03 1 1 4 4 54.5 43.9
03 1 1 4 5 2326 3368
03 1 1 4 6 08.7 09.7 07.6
04 1 1 4 1 068.9 070.5 071.3 070.7 072.0 070.6 070.6
04 1 1 4 2 16.8 19.4
04 1 1 4 3 34.6 37.8
04 1 1 4 4 51.6 45.7
04 1 1 4 5 3431 3068
04 1 1 4 6 12.5 12.8 07.8
05 1 1 4 1 076.2 077.9 078.3 076.3 077.8 077.5 078.5
05 1 1 4 2 14.8    -1
05 1 1 4 3 24.3    -1
05 1 1 4 4 54.3    -1
05 1 1 4 5 1415    -1
05 1 1 4 6 14.3 11.0 10.9
06 1 1 4 1 072.6 073.1 071.5 069.6 069.9 069.4 070.2
06 1 1 4 2 17.9 14.0
06 1 1 4 3 52.5 47.8
06 1 1 4 4 32.5 39.3
05 1 1 4 5 2217 3385
06 1 1 4 6 24.5 19.5 19.4
07 2 1 4 1 092.7 092.6 092.0 089.6 090.2 087.0 088.2
07 2 1 4 2 14.9 13.9
07 2 1 4 3 41.8 43.4
07 2 1 4 4 41.9 43.0
07 2 1 4 5 2142 1602
07 2 1 4 6 23.4 19.6 16.1
08 2 1 4 1 099.8 102.2 100.5 095.1 094.4 093.0 098.0
08 2 1 4 2 24.2 14.2
08 2 1 4 3 40.5 29.4
08 2 1 4 4 33.4 49.6
08 2 1 4 5 1916 1265
08 2 1 4 6 29.7 24.4 21.6
09 2 1 4 1 058.5 059.1 059.2 056.9 057.3 058.7 057.6
09 2 1 4 2 16.4 15.1
09 2 1 4 3 40.1 37.1
09 2 1 4 4 46.0 49.9
09 2 1 4 5 1948 2052
09 2 1 4 6 20.4 15.8 16.2
10 2 1 4 1 077.3 077.0 074.7 075.5 073.5 073.1 073.1
10 2 1 4 2 18.4 14.7
10 2 1 4 3 45.3 44.0
10 2 1 4 4 39.0 43.1
10 2 1 4 5 3002 5766
10 2 1 4 6 13.3 12.3 08.6
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11 2 1 4 1 061.7 060.5 062.2 061.2 061.8 062.4 061.7
11 2 1 4 2 18.2 16.4
11 2 1 4 3 44.1 52.5
11 2 1 4 4 36.0 34.0
11 2 1 4 5 3300 2768
11 2 1 4 6 13.4 11.0 10.1
12 2 1 4 1 082.5 081.2 079.4 079.5 079.9 078.2 080.2
12 2 1 4 2 17.7 14.6
12 2 1 4 3 53.1 41.6
12 2 1 4 4 31.1 45.4
12 2 1 4 5 2153 3759
12 2 1 4 6 18.2 12.8 12.9
13 1 2 4 1 088.4 092.0 092.3 093.0 089.8 087.5 087.0
13 1 2 4 2 18.1 18.3
13 1 2 4 3 52.3 46.0
13 1 2 4 4 31.4 31.2
13 1 2 4 5 4643 3294
13 1 2 4 6 12.1 11.5 10.1
14 1 2 4 1 076.2 076.8 075.1 077.0 078.4 076.6 077.4
14 1 2 4 2 14.7    -1
14 1 2 4 3 48.7    -1
14 1 2 4 4 34.1    -1
14 1 2 4 5 2169    -1
14 1 2 4 6 06.3 04.0 07.5
15 1 2 4 1 077.1    -1 079.5 077.6 079.5 079.5 079.8
15 1 2 4 2   -1 16.8
15 1 2 4 3   -1 52.4
15 1 2 4 4   -1 34.8
15 1 2 4 5   -1 2384
15 1 2 4 6 17.0 15.2 10.5
16 1 2 4 1 070.8 069.8 068.0 065.5 067.6    -1    -1
16 1 2 4 2 15.7 15.8
16 1 2 4 3 48.2 43.2
16 1 2 4 4 35.7 42.1
16 1 2 4 5 3296 3368
16 1 2 4 6 12.9 09.4    -1
17 1 2 4 1 058.3 059.3 060.0 058.9 058.3 059.7 058.9
17 1 2 4 2 14.4 15.7
17 1 2 4 3 46.0 49.0
17 1 2 4 4 41.2 36.3
17 1 2 4 5 1896 1816
17 1 2 4 6 16.0 16.9 13.5
18 2 2 4 1 080.7 082.4 081.2 082.0 081.7 082.7 080.3
18 2 2 4 2 16.1    -1
18 2 2 4 3 37.2    -1
18 2 2 4 4 41.7    -1
18 2 2 4 5 2334    -1
18 2 2 4 6 16.1 19.1 14.6
19 2 2 4 1 065.8 066.5 065.8 064.3 065.6 065.6 064.8
19 2 2 4 2 15.2 14.1
19 2 2 4 3 58.2 52.4
19 2 2 4 4 31.9 36.0
19 2 2 4 5 2553 3594
19 2 2 4 6 12.1 08.8 05.1
20 2 2 4 1 080.3 082.0 080.9 082.0 080.3 079.9 079.8
20 2 2 4 2 21.6 16.8
20 2 2 4 3 52.3 47.3
20 2 2 4 4 28.1 29.5
20 2 2 4 5 3577 2195
20 2 2 4 6 13.8 14.7 11.6
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21 2 2 4 1 064.4 064.8 064.3 063.9 063.2 061.5 061.9
21 2 2 4 2 14.8 16.0
21 2 2 4 3 48.3 42.8
21 2 2 4 4 48.3 42.8
21 2 2 4 5 2751 2339
21 2 2 4 6 09.1 09.0 03:8
```

Q-V SDH Activity

COLUMNS: 1 Group (1 = Exercise 2 = Control)

3 Time (1 = Pre-test 2 = Post-test 3 = Post-detraining)

```
1 1 2.86
1 1 3.65
1 1 8.20
1 1 3.80
1 2 7.73
1 2 7.57
1 2 5.75
1 2 6.85
1 2 4.30
1 2 7.30
1 3 1.55
1 3 1.36
1 3 9.30
1 3 3.40
1 3 6.60
1 3 3.26
2 1 5.57
2 1 2.91
2 1 8.30
2 1 1.54
2 2 6.41
2 2 4.53
2 2 3.92
2 3 2.20
2 3 1.20
2 3 2.16
2 3 5.50
2 3 4.60
2 3 3.82
```

Q-VI Fiber Types

COLUMNS: 1 Group (1 = Exercise 2 = Control)
3 Time (1 = Pre-test 2 = Post-test 3 = Post-detraining)
Variables = # fibers, % ST, % FTa, %FTb
(-1 = missing data)

```
1 1 365 49.9 44.7 05.4
1 1 253 59.7   -1   -1
1 1 092 68.5   -1   -1
1 1 285 54.4 34.4 11.2
1 1 161 59.6 26.7 13.7
1 2 232 38.4 48.7 12.9
1 2 303 47.8   -1   -1
1 2 113 38.9   -1   -1
1 2 189 40.7 56.6 02.7
1 2 221 49.8   -1   -1
1 2 264 55.3   -1   -1
1 2 142 62.0 31.0 07.0
1 2 204 81.4 18.6 02.4
1 3 196 59.1   -1   -1
1 3 239 65.7   -1   -1
1 3 236 41.9   -1   -1
1 3 201 39.8   -1   -1
1 3 385 60.2 31.2 08.6
2 1 215 60.9   -1   -1
2 1 245 54.3   -1   -1
2 1 148 63.5   -1   -1
2 1 200 50.5 36.5 13.0
2 1 452 40.0 30.3 29.7
2 2 125 51.2   -1   -1
2 2 197 66.0   -1   -1
2 2 185 67.6   -1   -1
2 2 149 74.5   -1   -1
2 2 185 42.2   -1   -1
2 3 298 67.1 28.5 03.7
2 3 308 55.5 28.9 15.6
2 3 255 54.9   -1   -1
2 3 159 62.9 22.6 14.5
2 3 255 64.3 31.0 04.7
2 3 326 71.8 21.8 06.4
```

APPENDIX R

TERMINOLOGY

anaerobic threshold (AT)

Onset of metabolic acidosis. (Davis et al,1976)

detraining

Cessation of formal training. (Drinkwater and Horvath, 1972)

endurance

The ability to persist in performance of physical activity.

fast twitch (FT)

Skeletal muscle fibers that stain dark for myofibrillar ATPase following alkaline pre-incubation.

lecithin cholesterol acyltransferase (LCAT)

Enzyme which catalyzes the conversion of lecithin and unesterified cholesterol to lysolecithin and cholesterol. (Lopez,1976)

lipid

Water insoluble biomolecule with high solubility in organic solvents. (Stryer,1975)

lipoprotein

Macromolecular complex of lipids and protein. (Lopez, 1976)

maximum oxygen intake ($\dot{V}O_2$ max)

The highest oxygen intake that the individual can attain during physical work breathing air at sea level.

metabolic

Referring to biochemical reactions which cause the formation of metabolites and the manufacture of ATP. (Stryer,1975)

myofibrillar ATPase reaction

Histochemical stain used to designate muscle fibers as FT and ST . (Houston,1978)

NADH-diaphorase reaction

Histochemical stain used to differentaite muscle fibers on the basis of oxidative potential. (Houston, 1978)

slow twitch (ST)

Skeletal muscle fibers that stain light for myofibrillar ATPase following alkaline pre-incubation.

succinate dehydrogenase (SDH)

Enzyme of citric acid cycle which catalyzes the oxidation of succinate to fumarate with the concomitant production of $FADH_2$. (Stryer,1975)

systemic

Referring to the flow of arterial blood from the heart to the body tissues and of the venous blood back to the heart.

ventilatory equivalent ($\dot{V}E/\dot{V}O_2$)

Ratio of volume of expired air to volume of oxygen consumed. (Davis et al,1979)